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THE BEHAVIOUR OF FREE HCL CURVE IN A SERIES OF ONE HUNDRED CONSECUTIVE CASES OF GASTRIC CONTENT ANALYSIS

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The present paper deals with the patho-physiological aspects of gastric content analysis in a series of 100 consecutive cases forwarded to the department from the hospitals. Our assumption is that all of these cases had some kind of dyspeptic symptoms for which they were subjected to a test meal examination.

Fractional analysis has been the only method of examination in all these cases after the usual oatmeal gruel. The fasting as well as the samples of stomach contents collected at quarter hourly intervals were titrated with N/10 NaOH for acid value and curves were plotted in charts to represent both the number of c.cm. of N/10 NaOH required and their corresponding value of HCl in percentage. These curves have been later subjected to a scrutinising study from many different points of view. The results of this study is given below.

When all the curves are viewed together these appear like a jumble of irregularities,—each curve differing from the other in many respects. A careful examination, however, shows that when they are superimposed one upon the other these curves can be classified into several broad groups which are typical.

Group I (24 cases) is characterised by a sharp fall of acidity in the first sample after the test meal. This is followed by an equally sharp rise of acidity which in a large proportion of cases exceeds the initial level.

Group II (31 cases) also shows a marked fall of acidity followed by a rise. When this is compared with the previous group the reaction appears to be slower. Consequently the angle between the falling and the ascending limbs is widened.

Group III (20 cases) is a curve of low acidity. During the dilution phase the acid value drops to nil and the subsequent rise in the later samples is both slow and small.

The Group IV (8 cases) is peculiar. It appears as a plateau like curve and does not show the usual effects of dilution. The rise, however, is not very marked.

The rest of the curves (12 cases) are irregular and may be looked upon as such.

An average curve from each of the groups mentioned above has been shown in Chart I, Figures 1—4.

CHART I. SHOWING THE DIFFERENT TYPES IN WHICH THE FREE HCL CURVE CAN BE GROUPED

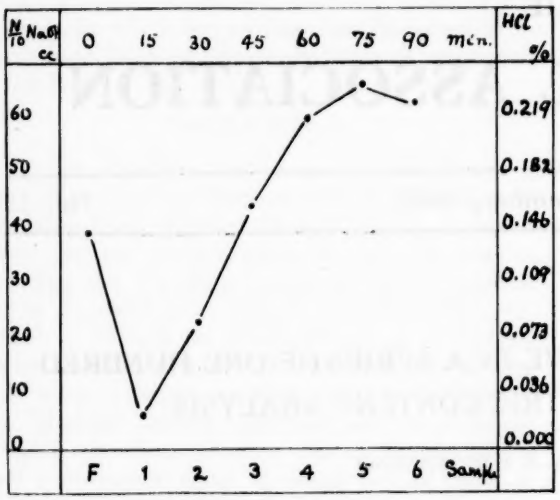


Fig. 1.—An average curve from Group I (24 cases)

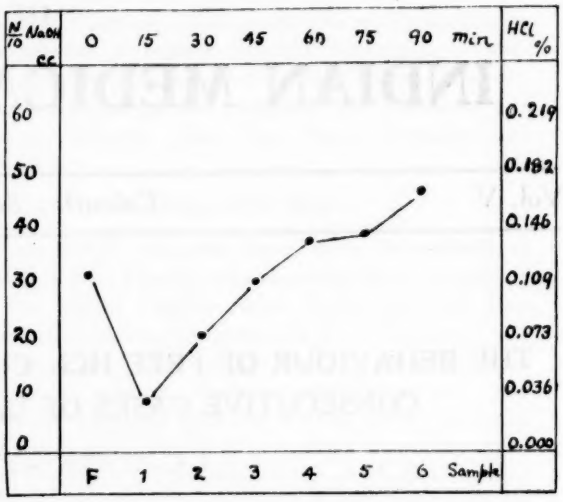


Fig. 2.—An average curve from Group II (31 cases).

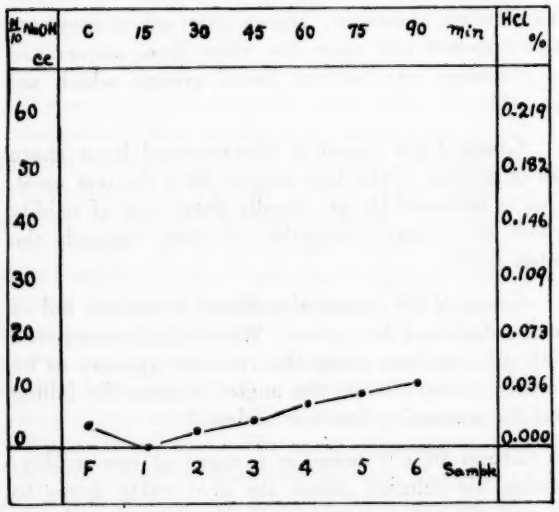


Fig. 3.—An average curve from Group III (20 cases).

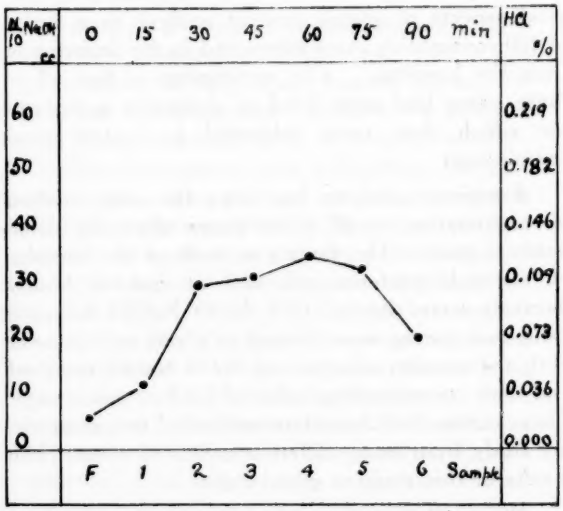


Fig. 4.—An average curve from Group IV (8 cases).

It is very difficult to say how far such a classification is helpful to us in the assessment of the results of gastric content analysis since we are dealing with cases which are assumedly abnormal and we lack a normal type for comparison. Still we have the impression that grossly abnormal cases are to be found in Groups I and IV while many of the normal cases must fall in the Groups II and III which are to be looked upon as less pathological.

In this connection it may be interesting to study the average curve obtained by taking all the 100 cases together. This curve compared with that obtained in the male subjects and the females separately shows a peculiar behaviour. (See Chart II, Figs. 1—3). There is a marked parallelism among the curves and the curve from the females has an continental figures seem to be rather too high for our purpose.

The grouping is as follows:—

Class I showing no acidity in the fasting sample 22
Class II showing an acid value below 0·073% 37
Class III showing an acid value below 0·146% 17
Class IV showing an acid value above this level 24

It will be seen from the above that the group of subacidity out-number the rest by far. This group together with the cases showing no acidity constitute the majority of our cases. The so called normal is but a minority only.

CHART II. SHOWING THE AVERAGE FREE HCL CURVE FROM ALL MALES AND ALL FEMALES AND FROM MALES AND FEMALES TOGETHER FOR COMPARISON. STRIKING PARALLELISM CAN BE SEEN. FEMALES HAVE A LOWER ACIDITY CURVE.

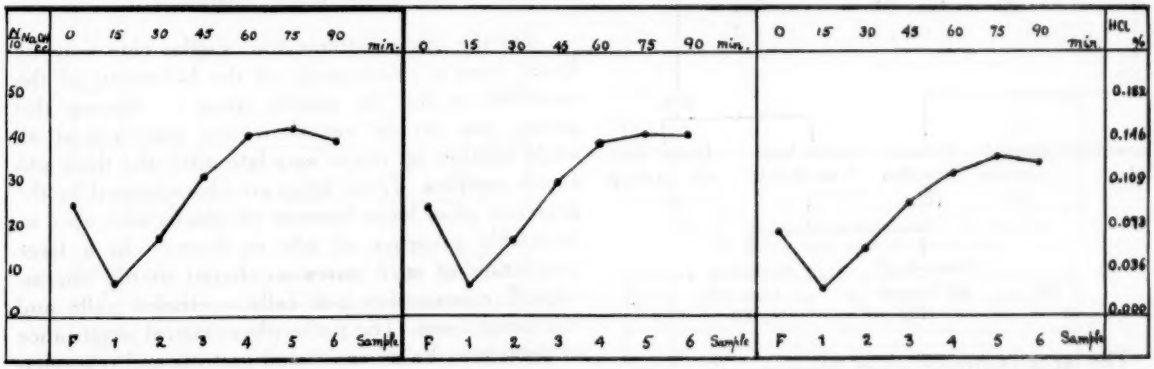


Fig. 1.—An average curve from all males (75 cases).

Fig. 2.—An average curve from males and females (100 cases)

Fig. 3.—An average curve from all females (25 cases).

uniformly lower range than in the males. The actual proportion of males to females in the series amounted to 75: 25. The generally held view that the acidity curve obtained in the females is lower than that in the males finds a corroboration here.

The next point we have tried to study was regarding the individual reactions: Taking the initial fasting acidity as the basis the series may be subdivided into four broad classes. In this classification we have taken the normal figures obtained by Bennet and Ryle as the standard. We have no standard normal figures for our country and the

These different groups of cases react differently in response to the test meal stimulus. The manner in which they react may be classified into the following types:

Type I showing no reaction to the completion of the test.
Type II showing only a sluggish reaction.
Type III showing the so called normal reaction.
Type IV showing a hyper-reaction.

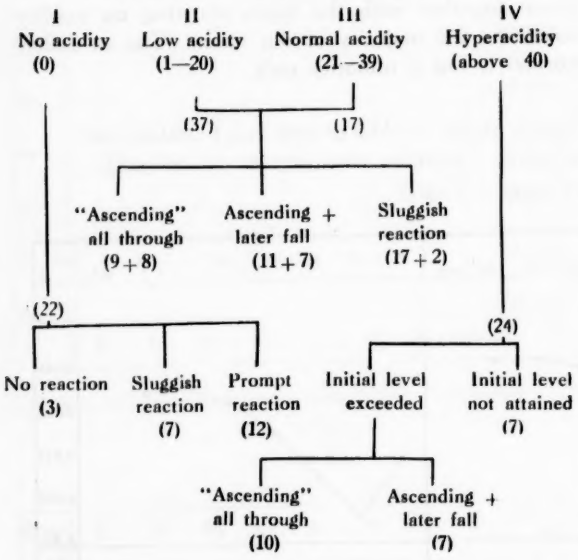
Here again we have adopted the normal type of reaction of Bennet and Ryle series as our

standard. Such normals have been described as "beginning with a low acidity it falls still very low in the first sample and rises subsequently to a figure scarcely higher than 40 c.c. N/10 NaOH. The later specimens show a tendency to fall."

The following Table gives in short our position with regard to the above classification as has been actually obtained in the analysis.

TABLE NO. I

THE BEHAVIOUR OF INDIVIDUAL REACTIONS ANALYSED. THE CLASSIFICATION IS BASED ON THE FASTING ACID VALUE. THE NUMERICALS INDICATE THE ACTUAL NUMBER OF CASES IN EACH GROUP.



The accompanying chart illustrates the individual typical reactions. (See Chart III).

Without clinical material to our aid it is very difficult to suggest anything regarding the significance which may be attached to these results. But as we have stated at the very outset, we have attempted to study this aspect of the question leaving the clinical study aside. Our results may be viewed in that light.

Gross abnormality is indicated in the extremes of the types, namely in the groups of 'No acidity' and 'Hyperacidity'. Although, according to the observations of Ryle and Bennet, normal subjects sometimes show these types of reaction, their percentage is but a negligible minority. Many serious conditions may be hidden in their folds: For instance, carcinoma, serious types of gastritis, pernicious anaemia in the first type and gastro-duodenal ulcer in the latter.

Further clue as to their true nature may be suggested from a closer scrutiny of the curves. Taking the hyperacidity group of curves, we find two different reactions: One we have designated as the 'ascending' type of curve. Here the initial fasting value is far exceeded during the course of the test. These cases represent not only a hyperacidity but hypersecretion in addition to this. This group must include the majority of duodenal ulcer with hyperacidity.

The second type is also an ascending reaction but differs from the above in that the initial fasting value is not reached at all during the whole course of the test. Our conclusion is that in these cases we have to think about some factors which will cause an accumulation of acid juice in the stomach as in the cases of obstructive dilatation.

On the other extreme too, similar clue may be found from a closer study of the behaviour of the reactions in the 'No acidity group'. Among this group, one set do not show any reactions at all while another set reacts very late after the third and fourth samples. These latter are characterised by the presence of a large amount of mucus and also an invariable presence of bile in them. In a large proportion of such cases we found in the microscopical examination pus cells, epithelial cells and microorganisms. The patho-physiological significance of these may be an associated gastritis and regurgitation of bile in an atonic stomach.

A third set in this group is more interesting. Here we find a very prompt reaction,—in some instances even the first sample has already shown the tendency of the acid curve to rise. The reaction following this is an ascending one. The height of the acid curve exceeds that of a normal and some even resemble the hyperacid group. In these cases the fasting sample shows the presence of stringy mucus and the juice is thick in consistency. The absence of acid in the fasting contents may be ascribed to an excessive secretion of mucus which has not only covered the surface of the stomach but has also hindered the secretion of the acid juice. As soon as the mucus has been washed off by the

CHART III. SHOWING TYPICAL INDIVIDUAL REACTIONS WHICH MAY BE OF CLINICAL Ssignificance.
(FOR DETAILS SEE TEXT)

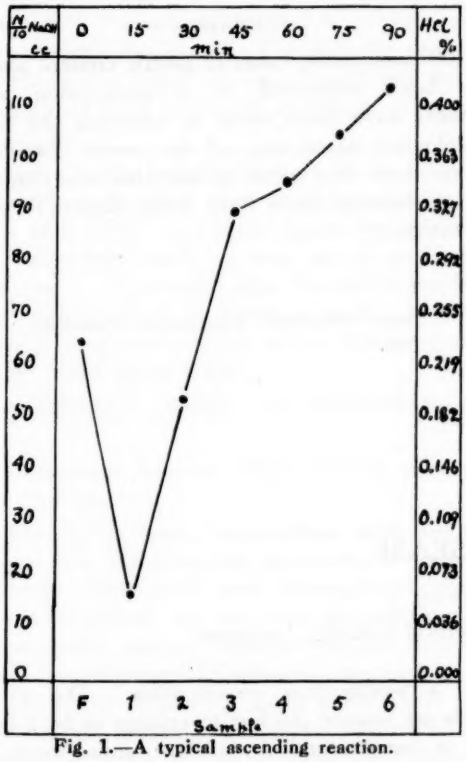


Fig. 1.—A typical ascending reaction.

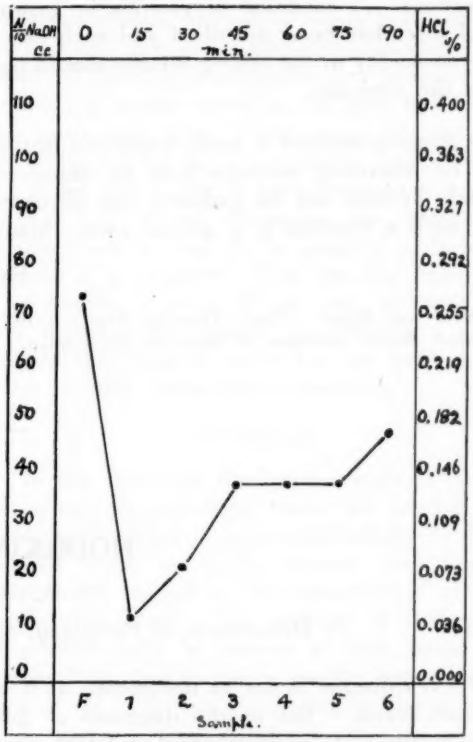


Fig. 2.—A typical curve where an element of obstruction may be suspected.

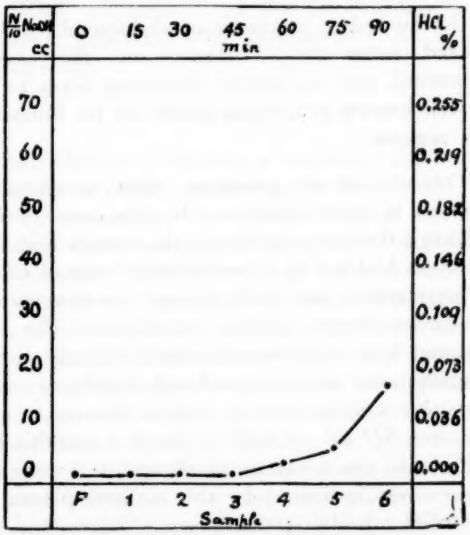


Fig. 3.—A typical curve from "No acidity" group showing a prompt ascending reaction.

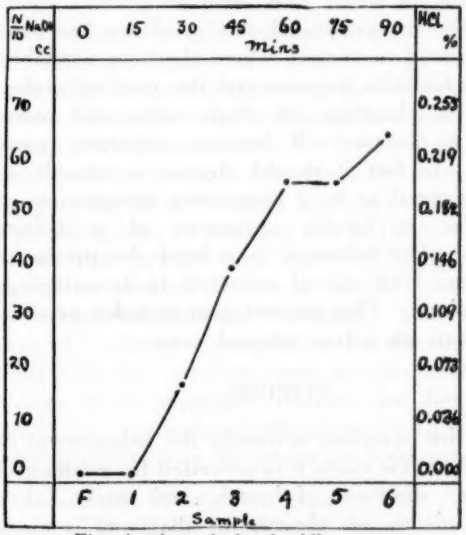


Fig. 4.—A typical subacidity curve.

oatmeal gruel the true nature of the reaction manifests and an ascending curve is the result. Both hyperacidity and hypersecretion is indicated in the later reactions. A condition of gastritis in gastro-duodenal ulcer has been described and such cases showing no acidity in the fasting sample should not misguide the clinician.

The sluggish reaction is partly explained in this light. The remaining reactions may be described as normal. It must not be gathered that all cases showing such a reaction is a normal case. Many factors complicate matters that are actually present. These have to be weighed and judged before anything definite can be said about them.

SUMMARY

100 consecutive cases of gastric content analysis have been subjected to a scrutinising study. Attempts have been made to ascertain the pathophysiological significance of the results obtained,—how far these may afford us any clue to a diagnosis. The outstanding facts have been shown in charts and tables.

REFERENCES

1. BENNET AND RYLE: Guy's Hospital Report. 1921. Ref. Recent Advances in Medicine, 4th Edition.

2. G. VON BERGMANN: Funktionelle Pathologie. Berlin, 1932.

HODGKIN'S DISEASE

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Hodgkin's disease is not so uncommon as it is usually considered. But as the diagnosis of this condition mostly rests on histological and biological examinations, and as the facilities for these tests are usually not within easy reach of general practitioners, the recognition of this condition has been mostly limited to centres where they are available. As these facilities increase and the medical profession takes advantage of them more and more, Hodgkin's disease will become relatively more frequent. In fact it should always be thought of when a general or local progressive enlargement of glands is not frankly suggestive of a definite diagnosis. The following is a brief description of this disease with special reference to its pathology and aetiology. This account also includes personal observations on a few atypical cases.

SYMPTOMS

The first symptom is usually the enlargement of glands. In a few cases it is preceded by swelling of abdomen,[1] shortness of breath, and rarely, when enlarged glands are absent, the diagnosis is made after a postmortem examination.[2] The cervical glands are usually the first to enlarge to be followed later or concomitantly by axillary, groin, mediastinal and abdominal glands. Rarely the enlargement may be limited to mediastinal, abdominal,[1] or other localised areas only. Pressure on the cervical, mediastinal, and abdominal structures leads to various well known symptoms produced by tumours in those regions.

Pressure on air passages, with accompanying dyspnoea is quite common. In one case[2] of acute Hodgkin's disease pressure on the vessels and active infiltration had led to a transudative type of effusion into pericardial and both pleural cavities with the consequent heart failure syndrome. The fluid contained large number of endothelial cells suggesting malignancy and accumulated rapidly enough to necessitate a daily tapping and to remove from the three sacs 527 oz. of fluid in about a month's time. In this case the invasion of pleural and pericardial surfaces was indicated by the marked pleural and pericardial rub after paracentesis. Spleen and often liver are usually enlarged. Peritoneal effusion may

be present. In one case pressure of the Hodgkin's tissue on bile capillaries had produced jaundice and the diagnosis was made after a post-mortem examination.[2] Pressure of the glands on the nerves leads to neuritic symptoms. Occasionally pressure of the Hodgkin's tissue on spinal cord has led to paraplegia and other signs and symptoms of tumour of spinal cord.[4]

Fever is a characteristic feature of the disease. It is usually of irregular type and the temperature may go upto 104°F.[1] or some times become subnormal[2] in severe cases. It may be of continuous typhoid type. A periodic type in which pyrexial and apyrexial periods of about a week's duration alternate is very characteristic of the disease and is known as 'Pel-Ebstein type'[1].

The following changes are observed in the blood:—

1. Progressive anaemia which may be of severe type in later stages.

2. Usually moderate leucocytosis with preponderance of polymorphonuclear leucocytes is present.[3] Occasionally leucopenia[2] with diminution in polymorphs is observed. In one case the polymorphs were completely absent.[1] Rarely a picture of acute lymphatic or mono-nuclear leukaemia appears in the last stages of the disease. A slight eosinophilia may or may not be present, though an eosinophilia is considered to be in favour of Hodgkin's disease. Radiological examination of the bones may show tumour-like deposits with osteo-clastic or osteo-plastic changes.

Diagnosis

When Hodgkin's disease is suspected the practical difficulty is usually to distinguish from a chronic inflammatory condition like tuberculosis on one side and from other malignant conditions especially lymphosarcoma. Clinically a generalised enlargement without any matting, a Pel-Ebstein type of fever, eosinophilia, and progressive anaemia are in favour of Hodgkin's condition. But a final proof is given by the histological examination of a gland or tumour tissue which shows the characteristic features described later. Recently Gordon has described a useful test[5] in the diagnosis of this disease. A 10 per cent. broth emulsion of a suspected gland is made under aseptic conditions. A rabbit is put under ether anesthesia and 0·4 c.c. of this emulsion is injected intracerebrally and 0·6 c.c. intravenously into the marginal vein of the ear. After an incubation period of 2 to 6 days, if the gland is from a case of Hodgkin's disease, the rabbit shows head-retraction, paresis, and fits but especially muscular rigidity combined with inco-ordination and ataxia, and a condition of spastic paralysis, but seen by observing their gait on the floor. These symptoms are invariably accompanied by a progressive loss of weight. The rabbit dies within 10 days (average) or gradually recovers. If these symptoms are present the Gordon's test is said to be positive. If nothing happens it is negative. This test has been found to be positive in about 75 per cent. of cases which were histologically positive for Hodgkin's disease. This encephalitogenic agent has not been reported so far in other pathological conditions.

Pathology

In one word the disease is a marked proliferation of reticulo-endothelial tissue and for this reason it has been called reticulo-endotheliosis by Piney.[7] As this tissue is mostly present with lymphoid structures it results in the enlargement of lymph glands and spleen. The increase of this reticulo-endothelial tissue is present in liver, lung, bone-marrow, intestine and to a smaller extent in other parts of the body too, depending on the amount of R. E. tissue in those organs.

The synonyms lymphadenoma, malignant lymphoma, and lympho-granuloma which depend on its association with lymphoid tissue are misleading from the pathological point of view. As the abnormal proliferation is limited mostly to the undifferentiated reticulum cells the term reticulosis has been coined by some American authors. Weber's Hodgkin's reticulo-mitosis is also suggestive of a malignant process. Hodgkin's disease is always characterised by pleomorphism of cells but one or the other types of cells may predominate; and Hodgkin's disease may be subdivided according to the predominant cells (endotheliomatous, reticular, cellular and sclerotic).[8] But as usually both reticular and endothelial cells are involved and as the proliferation behaves in a neoplastic manner the best name would be reticulo-endothelioma. The name Hodgkin's disease is of course non-committal.

It should always be regarded of malignant nature. The highly malignant forms have been

grouped under the term Hodgkin's sarcoma of Ewing. Gordon[9] considers that there appears to be a series of gradations between typical lymphosarcoma and typical lymphadenoma. Some American authors[10] have even gone further and say that Hodgkin's disease is genetically related to lymphatic leukaemia, lympho-sarcoma, and mycosis fungoides and that examples of mutation from the less to the more severe forms are constantly coming forwards. For example Hodgkin's disease has been known to change into lymphatic leukaemia and lympho-sarcoma, and Wile & Stiles have reported a case of mycosis fungoides changing into Hodgkin's disease. They like to include all these four conditions under the term lymphoblastomas—neoplasms of the malignant type. The question of their inter-relationship is in a melting pot, but it is hoped that their inter-relationship will be finally definitely proved and the present day confusion in the tumour pathology of lymphoid structure will disappear.

Macroscopically Hodgkin's tissue appears as whitish deposits in spleen and glands. Microscopically it consists of (1) endothelial cells with large neucleii (2) giant cell with one or more large neucleii (3) eosinophilic binucleate or mononucleate small cells (4) fibrous tissue (5) hyaline tissue representing degeneration and (6) hyperchromic and dividing nucleii characteristic of a malignant process are present in a more or less degree.

Eosinophils may be abundant (chronic cases) few or absent altogether (acute cases). According to one view eosinophils are derived from the metaplasia of R. E. cells. But it is not clear how could metaplasia of R. E. cells result in eosinophils only and not other types of granular cells. The other view that they are of haemic origin appears to be more plausible. They either represent the remnants of a chronic inflammation which excited the Hodgkin's process or accumulate, like lymphocytes in carcinomata, as a response to Hodgkin's tissue irritation.

Production of cellular and sclerotic types of Potter[8] depending on the variations in the amount of fibrous tissue also depend on the same unknown factors as in the productions of scirrhous and encephaloid cancers. The fibrous tissue is derived from the proliferation of the existing connective tissue and does not seem to be the result of metaplasia of R. E. cell as postulated by Potter.[8]

An interesting and plausible explanation of the pathology of blood changes in the disease depends on the increase of Hodgkin's tissue in the bone-marrow. In the beginning this tissue, like secondary deposits of cancer in marrow, acts as a sort of foreign body and irritates the marrow to pour forth more leucocytes into the blood streams resulting in a moderate leucocytosis of polymorphonuclear type. The process may remain constant at this stage. If the tissue increases leucopoiesis diminishes on account of the destruction or replacement of leucopoietic tissue. Moreover the reticulo-endothelium which is normally concerned in leuco- and erythropoiesis is becoming neoplastic and the proliferation is so rapid that there is no differentiation into leucocytes and erythrocytes. This results in a gradually increasing leucopenia and anaemia. The final stage may end in complete absence of granular cells and aplastic anaemia.

Aetiology

Though a lot of work has been done to elucidate the cause of Hodgkin's disease it still remains in dark. It has long been considered a sort of chronic inflammation as the name Hodgkin's granuloma suggests, but no definite cause of this inflammation has ever been found out. The view that it is due to avian tuberculosis has been practically definitely disproved. The present trend is to regard it a neoplasm. Gordon's discovery of an encephalitogenic agent in an emulsion of the glands lends a strong support in favour of some toxic or infective agent. So far no infective agent in the form of a filtrable virus or bacterium has been isolated. That the agent is not destroyed by alcohol or acetone and resists a temperature of 65°C. seems to indicate that it could not be a living virus or bacterium.[6] The idea of an infective encephalitogenic agent is also weakened by Friedman's discovery[11] that this agent is also present in normal bone-marrow, spleen, and leucocytes. Moreover the absence of any naked eye changes and only slight lymphocytic reaction in the brain of rabbit thus treated does not seem to support an infective theory.

Another view that this is a sort of bio-chemical or more precisely a proteolytic agent is gradually gaining ground and is more in conformity with the facts so far observed. Jochman showed that a proteolytic ferment is present in the bone-morrow,

spleen, and leucocytes—and not elsewhere—of man, monkey, and to a slight degree dogs.[11] And it is only bone-marrow, spleen and leucocytes which contain the encephalitogenic agent. With the knowledge that phagocytosis is the property of leucocytes and reticulo-endothelial cells, that phagocytosis is followed by lysis of the ingested cells and bacteria presumably through the agency of a proteolytic ferment, and that leucocytes and R. E. cells are mostly present in bone-marrow and spleen, it is in conformity with Jochman's discovery. And it is plausible to assume that through R. E. cells and leucocytes are found in other organs, the quantity of proteolytic ferment and consequently the encephalitogenic agent (if they are identical) is not present in a demonstrable quantity. With the extreme proliferation of R. E. cells in Hodgkin's disease the proteolytic ferment increases and leads to a positive Gordon's test through its lytic action on brain tissues. The lytic action of spleen, bone-marrow, and leucocytic extracts on brain tissue has been experimentally shown by Friedman.[11] But if the presence of an excessive amount of proteolytic ferment could be shown in Hodgkin's tissue the above theory would get another strong support in its favour. The lytic ferment is presumably not present in other neoplastic and granulomatous tissues and hence Gordon's test is negative, but this has also to be shown experimentally.

If the encephalitogenic agent and the proteolytic ferment prove to be identical the high hopes of solving the mystery of Hodgkin's disease fall to the ground, and its aetiology will still remain shrouded in mystery. The above view, if correct, would support the hypothesis that Hodgkin's disease should be included in the tumours. The neoplastic process starts, like other neoplasms, in a congenitally predisposed individual, the immediate provocative cause being some chronic infection leading to the enlargement of glands. As the cervical glands are usually the first to enlarge, septic teeth and tonsils appear to be the two most important exciting factors.

Prognosis and Treatment

This disease invariably leads to a fatal issue. An acute process may kill in a few months while a chronic one may take several years. As it is a neoplasm of the tissue which is widely diffused in the system surgical removal is out of the question. The time honoured administration of arsenic is practically useless. X-rays is the only therapeutic agent which is said to have some effect on this disease. It certainly produces some amelioration by its general property of inhibiting cell proliferation in localised areas. But it is easy to understand that a disease of such a widespread nature cannot be tackled by x-rays. The apparent subsidence is nearly always followed by recurrences and later wide dissemination of the process followed by the fatal end. Cures have been reported in very rare instances and it is likely that the process in these cases was only a hyperplasia of the reticulo-endothelial tissue and not a neoplastic one of Hodgkin's type. Of course x-ray will have to be used as a palliative measure and to relieve pressure symptoms and even to prolong life in some cases.

References

1. Gupta S. P.—*Ind. Med. Gaz.*, 1933 October, p. 573.
2. Burke & Hameed.—*Ind. Med. Gaz.*, 1935 March, p. 147.
3. Gupta S. P.—*Journ. Amer. Med. Assoc.*, 1935 I. 460
4. Wheatly—*Journ. Amer. Med. Assoc.* 1935.
5. Gordon—Br. *Med. Journ.*, 1933. I. 641.
6. Van Rooyan—*Br. Med. Journ.*, 1934, II, 519.
7. Piney—Recent Advances in Haematology 1931.
8. Potter—*Journ. Amer. Med. Assoc.*, 1935, I. 1858
9. Gordon—*Br. Med. Journ.* 1935, I. 532.
10. Wile & Stiles—*Journ. Amer. Med. Assoc.*, 1935, I 532.
11. Friedman—*Br. Med. Journ.*, 1934, II. 517

BIRTH CONTROL IN INDIA: NECESSITY AND METHODS

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PART I. BIRTH CONTROL AS A BRANCH OF PREVENTIVE MEDICINE

In this paper it is proposed only to treat Birth Control as a branch of preventive medicine and as to what methods the poor in this country can be taught. The well-to-do can afford to have any advice they care to have.

It is interesting to trace the history of the public health movement. In England which has always taken the lead in preventive medicine, there was no Public Health Act at all till 1848 and even then it was only partial and ineffective. In 1869, the Royal Sanitary Commission was convened by Parliament and as the result of its deliberations the Local Government Board was established in 1871 and the great Public Health Act which created Local Sanitary Authorities and made compulsory the appointment of sanitary officers was passed in 1875. Then for the first time the public health of the whole country came under regular medical supervision.

In the next 26 years much good work was undertaken. Schemes of water supply and drainage were initiated and housing was improved. The results of all these measures were shown in the improved death rates and lowered sickness rates. Paradoxically enough after 25 years work, statistics proved that there had been no decline in the infant mortality rate though there had been a marked reduction in the general death rate. It then became evident that the problem of infant mortality was not one of sanitation alone or of housing and that something more was necessary for its solution. Those concerned with the public health of the country began to hold that this loss of infant life annually recurring was largely the outcome of ignorance of mothercraft on the part of mothers. As a result, a movement was initiated early this century to provide trained and experienced women visitors to give advice to working class mothers as to the proper methods of rearing children. This is the beginning of the great maternity and child welfare movement.

The first work undertaken in connection with this was health visiting, then were started successively infant consultations, schools for mothers, antenatal clinics, and so on. Government helped these activities by passing legislative enactments like the Children's Act (1908), the Notification of Births Act (1915), the Milk Order (1919), and the Midwives and Maternity Homes Act (1926). All these measures and activities reduced the infant mortality rate from 150 to 75 per 1,000 births in about 25 years. The sponsors of the movement congratulated themselves on achieving such excellent results and felt satisfied.

So far much attention had not been paid to maternal mortality as it was considered the natural consequence of parturition, though maternal welfare work was also undertaken along with infant welfare work. In spite of careful antenatal work and improved maternity service, the maternal mortality, unlike that of infant mortality, was not showing any reduction. In this connection a well-known eugenist wrote to the writer of this paper: "Here in England, a good deal of maternal mortality is due to the fact that improved obstetrics covering now two generations are leaving each generation a higher proportion of women in the community whose pelvic formation is not suited to child bearing. Such groups were formerly eliminated by child birth owing to lack of proper obstetrical aid."

Then again all the specialised child welfare work that was being done was not leaving impression on the death rate of toddlers.

These were rather puzzling to the public health authorities. Even before this there was first a silent and then a vociferous demand for control of births though not at first focussed on medical or public health reasons. More and more light was being

thrown on hereditary factors by various scientific investigators. Then it was realised that not only the mother's health and enviroment but also hereditary factors and the income of the family played a part specially in infant mortality. The agitation for controlling births now assumed a scientific tone, as many doctors joined the movement.

In 1930, therefore, the Ministry of Health, England, gave permission to Maternity & Child Welfare Centres to give birth control advice to mothers in whom further pregnancy would be detrimental to their health and this was extended in 1934 to mothers on *all* other medical grounds as well.

Let us now review the situation in India. The infant mortality rate varies in different parts of the country from 200 to over 500 per 1000 births. On an average it may be put down to 200 to 250. If these figures are analysed, we find that very nearly 50 per cent. of these infant deaths occurred under one month and that 65 per cent. of the deaths under one month occurred during the first week of life which came to about 32 per cent. of the total infant deaths! It appears as if we are trying to make this world a breeding place for souls for heaven!

In India the first beginnings of Maternity & Child Welfare were made as far back as the 'sixties of the last century in the form of training dais, though organised work was started only in 1911, thanks to the efforts of Lady Willingdon who was then in Bombay. There is more or less a thoroughly organised and well conducted chain of welfare centres all over the country and yet in the last 25 years the reduction in infant deaths is almost negligible. Paradoxically enough, the infant deaths in rural areas where there is very little of welfare work and no sanitary measures are much less than in urban areas.

The maternal mortality rate in India varies from about 4 to 53 per thousand live births as compared with 4·0 in England and Wales. This rate also is not showing any reduction. The death rate of children aged 1 to 5 years in India is 66·3 per 1000 of the population of that age while in England it is about 16. Here also there is not only no reduction in deaths but in some places there is even an increase in spite of careful child welfare work! The conclusion is obvious, when we bear in mind that in our country the vitality of the people is poor and the expectation of life is small and that everyone marries irrespective of the fact whether he or she is biologically fit or not. The maternity service in rural areas is notoriously bad. In the first year of life, our infants get good nourishment without any cost to the parents because all mothers breast feed their infants. It is from the second year onwards that the father's poverty makes it impossible for him to give any suitable food to his children. It is the common experience of welfare workers to see a baby, a prize winner at the age of 9 months, looking a miserable skeleton with a bloated abdomen at the age of 18 months!

The following tables taken mostly from the U. S. A. Department of Labour will further explain the necessity for birth control.

TABLE 1

Interval between births	Infant mortality rate
1 year	147
2 years	98
3 years	86
4 years	85

TABLE 2

No. of Children in the family	Infant mortality rate
4 and less	118
6 and more	267
7 and more	280
8 and more	291
9 and more	303

TABLE 3

Order of birth	Infant motality rate
1st and 2nd born	138·3
3rd and 4th born	143·2
5th and 6th born	177·0
7th and 8th born	181·5
9th and later born	201·1

TABLE 4 (*Indian Figures*)

	Infant mortality rate	P. c. of still births to births	Children alive per 1,000 pregnancies.
5 Pregnancies and under —	226·3 ...	12·5 ...	633·7
6 Pregnancies and over —	272·2 ...	20·9 ...	504·4

TABLE 5

Earnings of father in dollars	Infant mortality rate
Under 400	170
450—549	130
550—649	120
650—849	110
850—1,049	90
1,050—1,249	70
1,250 and over	60

The infant mortality rate and the health of surviving children will thus be seen to be mainly dependent on:

(1) The mother's health.

(2) The child's heritage.

(3) The father's earning capacity.

(4) Child spacing and the number of children in the family.

In Russia when the economic condition was very bad, the Soviet Government legalised abortion, as they knew that people were already having recourse to it privately with disastrous consequences and that even the best known birth control methods were apt to fail in certain cases. We cannot go so far in India, because we have no trained maternity staff in rural areas except the proverbially unclean and untrained dai. There should however be available facilities for teaching birth control methods even in villages.

The objections usually raised in this connection are that the Indian women are either unwilling or unable to learn birth control methods. Though our women hate sterility, I can say from personal experience of them in urban and rural areas that they do ardently desire to be relieved of perpetual burden of child bearing. If this were not so, how can abortion or attempts at having abortion done be so rampant? The simpler methods of contraception to be described later can be learned even by the poorest and least intelligent women.

The other objection raised is that it will be impossible to carry the information to the 320 million people scattered over an area of 1,805,332 square miles in about 685,665 villages and 2,316 towns. If we propose to give instruction only in the most specialised methods and that only through lady doctors, then I agree that it is impossible, as there are only about 700 lady doctors in the whole country. The methods advocated for the poor are such that can be taught even to the village dais. If we can trust to the tender mercies of the dai, the life of the mother and the infant during the most critical period of their lives, why not entrust them with the simple task of imparting birth control information? They would, to begin with, be hostile to the movement and so were they when their training was started. A course of lectures on simple birth control methods should be given to every nurse, midwife and dai during their course of training and every district can now boast of training centres or classes for dais. It is much better to give a far greater number of women instructions in fairly efficient methods of birth control than to give the previleged few instructions in the most efficient and specialised methods. Propaganda among dais, men and women are required. That means demonstrations and talks on birth control should form part of baby week and health exhibitions.

The history of the public health movement is given in detail to show that birth control is but a natural sequence of the development of maternity and child welfare work.

PART II. BIRTH CONTROL METHODS FOR POOR INDIAN WOMEN

I do not propose to go into the specialised methods of birth control as these have to remain for many years yet the previlege of the well-to-do. An ideal contraceptive should have certain characteristics but here what we require is a fairly efficient contraceptive which is simple in use, easily available and cheap.

There are only two such methods, the medicated tampon and rubber sponge. The tampon is made of a wad of cotton wool or animal wool or rubber sponge or sea sponge. Among these the one readily available to Indian women is cotton wool. Surgical cotton should not be used but only that variety which is used for mattresses, etc. The cotton tampon should be, according to Dr. Dickinson, a ball of about 2½ inches in diameter or can be a flat dise of the size of the woman's palm and as thick as her thumb. As a general rule it might be mentioned that the bigger the tampon, even if it causes discomfort at first, the better protection it gives. Two tampons should be prepared and boiled in water. After squeezing out the water, both are put in a solution of lemon juice diluted

with equal quantity of water or in any bland oil such as cocoanut or sweet or neem oil. Take one out and insert it, in a squatting position, in the vagina as high up as possible, taking care not to squeeze out the solution. To facilitate removal, a thread or a small tape can be tied round it. After intercourse, the used tampon is taken out immediately. The other tampon should now be inserted in the vagina with two fingers as high as it will go. With a rotatory and outward motion, the vagina is swabbed out vigorously with it to remove every bit of the semen. The tampons are thrown away after use.

Sponges can be used in the same way. Sea sponges are useless for the purpose. A rubber sponge cut out to about the size of the woman's palm and ¾ inch thick is quite efficient. The rubber sponge can be used with the lemon juice or oils mentioned above. Dip the sponge in water, squeeze out the water, and dip in the oil, lightly squeeze out the oil and insert it as high as possible into the vagina. After use the sponge is washed, boiled and kept, as it can be used again and again. A contraceptive sponge can be cut out of the ordinary bath sponge, and so does not cost much. In Bangalore Indian made sponges are available at a cost of 4 annas each. Dickinson suggests also the use of the pulp of pomegranate and other acid fruits in place of lemon juice. Instead of cotton and rubber sponges, cloth, soft paper, etc. can be used as emergency measures.

It will be admitted that these two methods are simple to be taught even to dais and to be learnt even by unintelligent women. The tampon and the sponge may also be used with jellies. District board and Taluka dispensaries, if they are under the charge of enterprising doctors, can make their own jellies at a low cost. Dickinson gives the following directions for making a reliable jelly:

Starch-glycerate base.		
Boric acid	...	... 5 per cent.
Lactic acid	...	... 1 per cent.

Starch-glycerate is made with 70 parts of glycerine by volume, 20 parts of water and 10 parts of starch by weight. The glycerine is heated to a temperature of 125°C. to 130°C. The boric acid is allowed to dissolve in the glycerine while it heats. The water and starch are well mixed and when the glycerine reaches the desired temperature, the water and starch are added to the glycerine, while stirring hard and constantly until the mass hardens in a smooth starch-glycerate. The temperature is brought down to 70°C. during which time the mass is stirred occassionally to prevent the formation of lumps. At this temperature the lactic acid may be safely added and the material placed in the filler to be put into tubes.

The only other method that need be mentioned here is sterilisation, namely, salpingectomy and vasectomy. Salpingectomy means the removal of a small piece from each of the two fallopian tubes and ligaturing the cut ends. The usual operation is through the abdominal route, but it can also be done through the vagina. Whatever route is adopted, the operation is serious and should not be undertaken lightly.

Vasectomy, on the other hand, is a simple operation which even general practitioners can be trained to perform. The simplest procedure is the following:—

Sterilise the skin as usual, make the assistant locate the vas and hold it just under and against the skin. An inch of the area is anaesthetised by some local anaesthetic. The site selected is about half way down the cord. A small incision of about half an inch is made and the vas to which the attendant has been holding on is hooked out. By this method there is no need for hunting for the vas after the incision is made. The vas is now dissected out and about half an inch of it is cut out. For contraceptive purposes it is enough if the upper end is tied. If the lower end is tied, the testicle feels tender for some weeks because of the damming back of the testicular secretion. If the lower end is not tied, the testicular secretion passes into the loose tissues of the scrotum from where it is absorbed.

After the wound is dressed, the patient can go for work, though dressing has to be continued for a week or so. It is advisable to use a suspensory bandage for some time. The first three or four intercourses after the operation should be done with the aid of a sheath and the last discharge examined for the presence of spermatozoa. This is necessary because there might have been some spermatozoa in the seminal vesicles which should come out. When the semen is proved to be sterile,

all contraceptives can be given up. This operation is what is known as sterilising without unsexing. There is no lack of desire or of erection and no other change in the sexual powers after the operation. The quantity of the semen discharged also remains more or less the same, as the spermatozoa form only a small fraction of the seminal fluid. The patient does not suffer in general health also in any way. I have heard patients saying that they never enjoyed sexual intercourse with such freedom from anxiety as after the operation.

Women should also be taught not to put faith in the safe period or prolonged lactation.

References

1. George Newman.—An Outline of the Practice of Preventive Medicine.
2. R. L. Dickinson.—Household Contraceptives.
3. Michael Fielding.—Parenthood—Design or Accident.
4. P. K. Wattal.—The Population Problem in India.
5. Sir Arthur Newsholme.—Vital Statistics.
6. A. P. Pillay and M Subramanyam.—Maternity and Child Welfare.
7. A. P. Pillay.—Welfare Problems in Rural India.
8. A. P. Pillay.—The Medical Profession and the Birth Control movement (*Indian Medical Gazette*, Vol. LXVIII, No. 1).
9. A. P. Pillay.—Contraception in General Practice, (*The Medical Bulletin*, Vol. III, No. 22).
10. Marie C. Stopes.—On Some Aspects of Contraception for Indian Women, (*Marriage Hygiene*, Vol. II, No. 1).

NOTES ON PLAGUE

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PART I

Clinical Features

The following types of plague are well known:—

1. Bubonic consisting of inguinal, axillary and cervical types.
2. Septicaemic.
3. Pneumonic.
4. Intestinal. To these another type should be added:
5. Skin or carbuncular plague.

As regards axillary type of bubonic plague, Dr. Gaud[1] reports that affection of thoracic gland is most frequent and is found in about two-thirds of the cases. The axillary glands can be divided into 5 sub-groups—(a) axillary proper at the dome of axilla, (b) brachial, (c) sub-scapular, (d) sub-clavicular, and (e) thoracic. The upper portion of the thoracic sub-group is situated at the level of the 2nd and 3rd intercostal space on the thoracic wall of the axilla under the tendon of pectoralis muscle. The lower portion of the thoracic sub-group is situated at the level of the 5th intercostal space. Three points are brought forward in the same paper:—(1) Thoracic sub-group is most frequently affected in plague and brachial in common affections. When thoracic sub-group is affected, the tendon of Pectoralis is raised up forwards. This gives rise to sub-pectoral bubo. This is a diagnostic point in plague. (2) Generally one sub-group is affected in plague—so to say, plague separates the subgroups better than an anatomist. (3) Thoracic sub-group is connected with lymphatics of lung and pleura and so axillary plague gives rise to secondary pneumonic plague.

Skin plague has been described by Choksy[2]. Phylectenules, pustules, bullae or umbilicated bullae may be found in the beginning. Then necrosis supervenes and spreads. A line of demarcation appears. Eschar is greenish, cold, hard leathery, and depressed in the centre and firmly bound down to the tissues underneath. Skin around is red and studded with vesicles. Small and secondary necroses may be present. If buboes are present, they are mostly secondary. Course of disease is two to three weeks. Necroses have been observed on scalp, neck, chest, face, arm, forearm, shoulder, abdominal wall, lumbar and gluteal regions, vulva, scrotum, thigh, leg and foot. Destruction of tissues may involve large cavernous cavities, exposing bones after destruction of periosteum. Cartilages of the ear, eyelids and nasal bones may be involved.

Testes may be exposed if scrotum is involved, and intestines if abdominal wall is involved. If necrosis is small, bubonic features predominate. If necrosis is large, buboes are of subordinate significance. There is prolonged fever.

Bacteriology

Cultural characteristics are well known. Benzonova[3] reports that out of 150 strains which had been kept at room temperature for five months, four strains gave rise to uniformly turbid growth in broth. Much attention has been paid to distinguishing B. pestis from B pseudo-tuberculosis rodentium. (Table 1 & 2 appended).

Michelleti[4] reports that for examining a decomposing rat fix spleen smears in methyl alcohol and stain with alpha-naphthylamine Cultures may be made from bone marrow in preference to those from spleen.

Fusca[5] describes procedure for diagnosis of plague in rats. Rats dying of plague lose notion of direction and danger and die outside the holes. In newly dead rats the limbs are drawn, claws are clenched, like fists, head flexed on trunk. With passing away of rigor mortis, this attitude disappears. The first step—Do autopsy and examine glands particularly cervical, and spleen as well. Second step—Make a microscopic preparation of spleen juice, fix with methyl alcohol and stain with methylene blue. Bipolar staining will appear. B. pseudo-tuberculosis rodentium can be seen and this fact should not be neglected. Third step—Even when microscopic examination is negative, make culture from bone-marrow of a long bone in broth. Bacillary masses fall to the bottom. In these masses bacilli have the appearance of long chains, which do not show involution forms for several days. Fourth step—Test production of nitrites. Plague bacillus produces nitrites. Fifth step—Inoculate culture in the internal angle of the right eye of a guineapig. After two days the head of the guineapig turns on one side. This sign is not always present, but is characteristic. After 24 hours of inoculation plague bacilli are found in the purulent liquid of the eye. On the fourth day, animal dies and characteristic lesions of plague are found.

Antiplague serum was investigated by Indian Plague Commission.[6] They investigated opsonins and lysins. Greval and Dalal[7] investigated agglutinins and complement-fixing bodies in high titre sheep sera. For preparation of antigens and other details, original paper should be consulted. Result of testing agglutination of antiplague high titre serum against plague bacilli was as under :—

1 : 2000	...	1 : 4000	...	1 : 6000
+ +		+		+ −

Agglutination was tried against B. avisepticus and B. lovisepticus and B. pseudo-tuberculosis rodentium. The first two failed to show agglutination in any high dilution. The last also failed in 1 : 4000 and 1 : 6000 dilutions. Agglutinin absorption test distinuguished B. pseudo-tuberculosis rodentium. Complement-fixation was also tried and complement-fixing bodies found in the anti-serum. Agglutinating and complement-fixing bodies are separate and authors note that it will be interesting to find which of these bodies gives indication of immunity.

Vaccine Preparation

Several methods have been used for vaccine preparation—Agar culture vaccine, Neucleo-protein vaccine and Avarisphage vaccine have been tried. Phage-lysed vaccine has also been suggested by Flu[8] through experiments. Haffkine's method of broth vaccine is well known. Schutze[9] found that when cultivated at 37°C. plague bacillus develops a gelatinous capsule and Buchanan[10] suggested that Haffkine's vaccine be improved by culture at 37°C. as the gelatinous capsule will be good for producing immunity. Taylor[11] maintains that plague bacillus loses virulence at 37°C.

Standardization of Vaccine

Naidu and Jung[12] describe one method as follows :—Give ·5 c.c. or one-eighth of human dose. After 7 days infect them with spleen of a rat dead of plague (dose ·003 mg. subcutaneously). Find out number of survivors after 15 days. Vaccine is not of proper strength if less than 30 rats survive. Madras rats are susceptible so they are used for testing.

In the above method, number of germs in the spleen is undetermined, and so Sokhey[13] has elaborated another method of standardization. Take 10 white mice weighing 20 to 30 grms., inject 1/32 to 1/10 c.c. of vaccine and after ten days test for immunity. As test germs use 1·5 c.c. of 1 : 5000,000 dilution of 48 hours culture. The 1·5 c.c. of test dose should contain 30 to 100 germs. The strain of germs should be of such virulence that 3 to 10 germs will kill 80 to 100 per cent. of mice. Animals under test should not die within 21 days with plague.

Naidu and Jung[14] tested several brands of vaccines in 1929 and none was found better than Haffkine's vaccine.

A question which crops up frequently is whether there is any negative phase after anti-plague vaccination. Kapadia and Stevenson[15] found that in experimental animals, there is no negative phase even 1½ hours after injection of vaccine. Immunity begins to appear a few hours after injection, and goes on increasing for 2 to 3 days remaining strong even 14 days after. Kapadia and Stevenson[16] found that toxicity and potency are related but do not run entirely parallel. By storage for one year toxicity is reduced but not the immunising power of the vaccine.

Bacteriophage

Bacteriophage has not been found useful experimentally by Advier[17] and Pais[18] found it causing toxaemia and liver degeneration.

Inset Vector

Indian Plague Commission[19] had experimented with transmission of plague through bed bugs. Novokovi and Lalazorv[20] report successful transmission of plague through bed-bug. Experimentally many bed-bugs do die on account of infection. Do Radt[21] had succeeded in infecting animals through emulsion of head louse removed from a plague patient. Yamada[22] succeeded in transmitting plague through mites experimentally. Of course the fleas are the chief vectors; and *xenopsylla cheopis* is the most important in India. Many

flea surveys have been made to prove the importance of *X. cheopis* in plague epidemiology. It has been proved that plague is connected with 1 per cent. cheopis index with about 75°F. temperature and 1 to 10 millibars of vapour pressure deficiency.[23] These conditions are favourable factors for spread of plague. Astia index of seven per cent. may also be favourable for spread of plague.[24] Strickland and Roy[25] found that *Astia* is the chief flea of Calcutta and so Calcutta has escaped plague as compared with Bombay. Rat flea surveys of Madras Presidency by King, Iyer[26] and others lead to the same conclusion. Effect of temperature and vapour pressure deficiency do not affect all fleas in the same way. King and Iyer[27] report that *Astia* increases first from July to August; and then *cheopis* increases, and on onset of cold weather increases very greatly. *Cheopis* is more affected by seasonal variations. Pandit, George and others[28] report about the flea survey of Ootacamund. Plague epidemic in the Nilgiri district shows no marked variation between one season and another; while the death rate is highest in October to December, and least in March to April. *Ceratophyllus fasciatus and X. cheopis* both are found. Optimum temperature for transmission of plague through *Ceratophyllus* is 15° lower than in the case of *X. cheopis*. Thus favourable conditions for transmission of plague exist throughout the year.

Importance of *X. cheopis* leads one to conclusion that there must be some control of merchandize from *cheopis* area. This is of course very difficult.

Life history of flea is well known. Webster[29] could not distinguish between larvae of *cheopis* and *astia*. Life history of *cheopis* is affected by physical conditions. According to Hirst[70] the simplest measure of effect of climate in relation to the probable plague incidence is egg-laying ratio. Effect of good oviposition will be apparent after three weeks. Average longevity of unfed fleas is perhaps more sensitive to increasing vapour pressure deficiency.

De La Reu[31] reports an epidemic of plague in Morocco due to human flea. There the number of human fleas infesting houses is incredible. The shortness of the time this flea remains infective is the reason why plague in certain parts of Morocco is severe and then disappears completely.

Jumping power of flea is 4½ inches and climbing power 18 inches on moist glass[32].

Rodent Reservoir

In other countries susliks squirrels, marmots, tabargans, gerbilles and multi-mammate mice are the reservoirs. Mc Roy[33] reports that the disease carried from squirrels is milder. Rat is the chief reservoir in India. Rat gets immune in affected places and according to Mackie[34] this immunity is transferred hereditarily to their offspring. Thus rats of infected place become immune. Dorenbis[35] suggests that when plague bacillus and bacteriophage both are fed to rats, plague bacilli and bacteriophage both are found in rats. The flea which will ingest the blood of such a rat may become a vaccine centre as the rats to which it will transfer infection may become immune. Acute and chronic plague in rats are well known. We should add another type—plague without lesions (Williams and Kremmed 1923).[36] Long[37] in Peru also found presence of plague without lesions. Varas[38] describes many kind of lesions in rat plague. Suppurating glands were found without lesions and bacillus pestis in organs. One animal looked healthy upto 18th day of captivity and then showed plague on autopsy after sudden death.

Perpetuation of Plague among Rats[39]

There is no epidemic in off season due to diminished rat population, increased immunity in rats, high temperature and diminished number of fleas. Chronic rat plague is not important in perpetuation of plague as rats seem unlikely to contract plague by feeding on dead rats; and it is unlikely that chronic plague may light up. We shall see how plague is perpetuated in other countries. In California Mc Roy found acute disease in the squirrels throughout the year. In South Africa chronic plague is not found and in the off-season the disease is of a smouldering type *i.e.*, few animals are attacked. In some of the countries, rodents go to sleep and they wake up after long time with bacteraemia. To cite one experiment, Teh infected 15 tabargans. One tabargan did not sleep and died on the 6th day. 14 sleeping tabargans died after 88—130 days with bacteraemia. To search if fleas carry over plague to the next season, Jamonsky and Pollisk[40] collected 1799 fleas from suslik nests in off-season and found 5 of these giving positive cultures. One concludes as follows:—

(1) Chronic plague has not much significance in spreading plague.

(2) Bacteraemia in rodents is essential for spreading plague.

(3) Cases of carriers with bacteraemia may intervene but their role is not well understood.

It seems very important that search should be made for rat mortality during the off-season. Of course this is a bit difficult.

Hypothesis of Kunhardt and Chitre[41]

Mostly plague infection dies out but persists in a small number of villages of any size and in any situation. Persistence depends on a sufficient number of rats. What numbers will be sufficient depends upon the size of the town and the date when plague began. Villages can be classified into following four groups:—

(a) Villages almost certain to carry over plague. (b) Villages which will probably carry over plague. (c) Suspected places which require observation. and (d) Village unlikely to carry over plague. Class (b) contains villages with the following population and the period of beginning of plague:—

Over	25,000	population	and	plague began	before December.
Over	12,000	,,	,,	,,	in mid-December.
Over	42,000	,,	,,	,,	in mid-January.
Over	2,000	,,	,,	,,	in mid-February,
Over	850	,,	,,	,,	in mid-March.
Over	350	,,	,,	,,	in mid-April.

By taking this hypothesis into consideration, about 10 per cent. of villages can be selected for rat destruction; and thus money saved. Formula of Kunhardt and Chitre can be graphically represented as well.

Strickland[42] working in Belgaum and Dharwa districts found that formuls of Kunhardt Chitre did not quite suffice. He also stresses the consideration of the effect of grain trade and other traffic. Local conditions were somewhat different in Belgaum and Dharwar. Formula of Kunhardt and Chitre should be modified in light of local experience, and local seasonal variations. And then it seems that it shall prove useful.

Prevention

Means of prevention are well known. Anti-rat campaign can be most efficiently carried out by cyano-gasing. George and Webster[43] report that they gassed 110,000 holes. They succeeded in plague prevention. Snakes were also killed. One advantage of gassing is that fleas are also killed; and fleas probably take part in perpetuation of plague, and thus this is a great advantage. During actual outbreak of plague advantage is obvious.

Disinfestation of grain is a difficult problem. Pandit[44] found that sunning of bags is useless. Sulphuration of grain according to a report in 1932 spoils grain[45].

Treatment[46]

Surgical treatment consists of symptomatic and disinfectant treatment. Among disinfectants carbolic acid, mercurochrome, iodine and Bayer 205[47] have been used. But medicinal treatment is not satisfactory.

Vaccines and serum have also been used. Serum treatment has not given encouraging results. Serum is found to modify the disease, life is prolonged, septicaemia is lessened and mortality is not reduced to any appreciable extent. Vaccine in doses of 100 million germs has been tried. Row introduced staphylococcus pyogenes into bubo and claimed beneficial results. Bacteriophage has not been found useful.

Discussion

Gaud's observations on sub-pectoral bubo seem to be correct, within my experience.

I wish to draw attention to the climbing power of the flea. In village Chak Darshan I found four fleas on the patti of the coolies leg. Coolies had been, of course, beforehand taught to recognise and kill the flea with kerosene oil. As I killed the fleas one by one I saw them climbing over coolies patti. I would suggest trial of leg guard with sticky substance outside to give greater protection to the leg of the coolie. As regards perpetuation of plague I have mostly found that where plague appeared without importation rat mortality had been noticed by the villagers during the off-season, but villagers had given no importance to it.

In use of barium carbonate for rat-destruction often cats are found dead, presumably through eating poisoned rats. Dead jackals are also found presumably due to eating poisoned cats. Cat-dying is a disadvantage as cat is a rat-killer. This disadvantage is not present in gassing.

Table I[48]

Differentiation between B. Pestis and Allied Bacilli

	B. pestis	*Pasteurella*	*B. pseudo-tuberculosis rodentium.*
Growth in bile-salt medium	+	−	+
Indol and H_2S	−	+	−
Motility	−	−	+ below 30°C
Litmus milk	Neutral	neutral	alkaline
Rhamnose	+	−	+
Glycerol	+ −	−	+
Saccharose	−	+	−
Sorbitol	−	+	−
Virulence for white rats	+	+ −	−

Table II

Differentiation of B. Pestis and B. Psuedo-tuberculosis rodentium

Medium	*B. pestis*	*B. pseudo-tuberculosis rodentium*
3% salt and agar.	Involution forms.	No involution forms
10% salt agar.	Growth not suppressed.	Growth suppressed.
Malachite green broth.	Decolorised slightly and slowly.	Decolorised rapidly
Glucose peptone water.	Not made alkaline.	Made alkaline.
Maltose peptone water.		Fermented more rapidly.
Thymol blue sugar-free broth.		Greater alkalisation
Hungry acid agar[50] (without broth).	No Growth.	Grows.
Methylene blue in[51] broth (pH6·8—7).		More rapidly and intensely decolorised.

Table III[52]

Recognition of Rodents

Rodent	*Length of body and head (A)*	*Tail length (B)*	*Noise*
Rattus rattus	6-8	125	A series of squeaks
Rattus norvegicus	about 10	90	A prolonged squeak
Bandicota	over 9	90	A snarl.
Gunomys	less than 9	16-8	A grunt.
Neosakia	,,	8	—

A. Length of body and head in inches

B. Length of tail as percentage of body and head.

PART II

PNEUMONIC PLAGUE

Clinical Features

Prodromata are rare. The disease begins suddenly often with rigor, but marked chills are not the rule. Rise of temperature is slow. Temperature rarely rises above 103 (39·5°C.) though 40 to 40·5°C. have been recorded. Temperature drops to normal before death. In exceptional cases there may be no fever.

Early stage:—There is rising temperature and headache, pain in the limbs, general indisposition, weakness and slight vertigo and oppression in the chest, may be present. The skin is dry, face flushed, conjunctiva suffused and tongue coated. Nausea, vomiting, drowsiness, excitement, and delirium may be present. Weakness and prostration may be extreme. Generally marked prostration is absent in the early stage. Respiration is increased though the fact may not easily be perceptible. Respirations generally number 25 per minute. There is some dyspnoea which tends to increase. Lung signs are few, and less entry of air with few moist sounds at the bases may be found. Later there is dullness. Pulse deteriorates in frequency and quality and may be the first sign of serious disease.

Second stage:—Cough is first dry and then soon there is sputum. Attacks may be short or of long duration. Patient has no difficulty in emitting sputum. In a few cases, sputum is scanty and there is pain in the chest. Towards the end the patient is exhausted to bring up much sputum. This is characteristic of pneumonic plague. Sputum is at first salivary and then blood begins to appear either in streaks or more mixed and rusty. Sputum may also be copious and haemorrhagic. In early stages B. pestis may be absent or else it is easy to diagnose through sputum examination. Towards the end, the patient gasps for breath and pulse may not be felt at all. Deep cardiac dullness is increased in area and secondary pulmonary sound is accelerated. There may be diarrhoea. Uterine haemorrhages may be present. Haffkine records a child birth in pneumonic plague. B. pestis was found in the heart blood of the child. Marked disproportion between serious general condition and the lung signs is the guide in diagnosis from pneumonia.

Epidemiology

Incidence in India:—Pneumonic plague epidemics have been reported. Gill reported that pneumonic plague in India is generally associated with bubonic plague and rat mortality. Gill described an epidemic of pneumonic plague which occurred in February, 1920 in which 1200 people died in a population of 6000 in a village in the Punjab.

Climatic factors:—Generally observers are agreed that the pneumonic plague occurs in very cold places; but in many instances it has spread in summer. Thirou[54] and Boye[55] report that in Madagascar pneumonic plague has been observed where minimum temperature is below 160°C. and has maximum frequency where temperature descends below 140°C. Teague and Barber assumed that low water deficit of the atmosphere cause droplets to flow about in air. Against this is the fact that pneumonic plague is infective in the vicinity only.

Infectivity:—Bacilli are ejected in coughing even when drops are not visible. Patients could eject bacilli upto 3½ft. There may be danger in patient's forcible speaking. Infection may take place through a short contact. One doctor contracted infection from a servant when he came close for a moment. Usually there is not history of long contact. In the first stage the patient is not infective. It should be the duty of all to isolate the patient within 24 hours which is the period of non-infectivity. There is also possibility of infection by floating droplets and also from rooms occupied be patients. In sunlight plague bacilli are destroyed soon in sputum; but in shade they are resistent. They survive in apparently dry sputum. Clothes and bedding may also spread infection. Opium smoking pipes have also been incriminated. There is also possibility of infection through corpses. (This point was stressed before Indian Plague Commission). Of course the most important spreading agent is the patient. Bacilli were found 5 months after burial in European cemeteries.

Dogs, pigs, cats and donkeys also get affected in pneumonic plague. Nikanorff[56] reports that spontaneous plague occurs in camel in Turkistan and in one case those who cut up the camel got infected.

Human carriers:—Existence of healthy and incubation carriers is doubtful. Incubation carriers seem to exist. Among convalescent carriers cases have been found where bacilli have been found present for 70 days after complete subsidence of lung signs.

Origin and decline[57]:—Origin generally lies in secondary lung infection in bubonic cases. Rise *de novo* is unlikely though some observers maintain that pneumonic plague may be contracted from rodents.

Decline is helped by hygienic measures; but question also arises as to how the disease declines naturally. In Harbin and Vladivostok the decline was due to increased virulence of B.pestis. Virulence became much increased and patients died soon. The patients were infected undoubtedly through respiratory tract; but on examination of cadaver, only hyperaemia and oedema were found in lungs. These cases were not much infective. A Russian peasant got shivers and died next morning. On post mortem bacilli were found in lymph spaces of adventitia and not in the blood vessels.

Experimental Production of Pneumonic plague.—Teh and Jettmar tried it and found that pneumonic plague invades the body through lower portion of the respiratory tract. String and Tengue infected monkeys through spray. The monkeys got pneumonic plague. Alterations in fauces and cervical glands were very slight.

Prevention.—Protective power of vaccination is much less than in bubonic plague. Experimentally it has not proved successful. Combined vaccine and serum therapy may be tried

for prophylaxis. The contact should be watched longer as such therapy prolongs the incubation period.

Nicolle[58] and others tried inhalation of 3000 million B. pestis besides subcutaneous injection. Authors recommend further trial. Scut[59] tried injections of Omnodin, a compound sarcine vaccine, containing albumin, lipoids and fats. He claims to have found it useful.

Teh's mask. Take a piece of gauze 3ft. × 9″ and a piece of cotton 4″ × 6″ and 1″ thick. Double the strip of gauze, keeping cotton in the middle. Make two cuts of 15″ at either end. Tie upper two ends at the back of the head above the ear and the lower ends below the ear. Of course the piece of cotton will be over the nose. Mask should be carefully adjusted and space beneath the nose should be filled with cotton if necessary. Dr. Young died due to neglect of this precaution. When seeing patients, a hood should be worn with a mica piece for the eyes and a piece of silk 4″ × 6″ over the nose besides the mask above described.

Further precautions are necessary for those who see patients. Washable overalls, rubber boots and gloves are also necessary. Thymol lotion 1 per cent. is good for gargling.

Disinfection.—Shut up house for a while for allowing droplets to settle. Cover floor and contaminated article with quick lime or spread saw dust and burn. Floor walls and furniture may be disinfected with liquid disinfectants. Formalin may be used where advisable. Coins found with corpses should be disinfected. Dead bodies should be disposed of by burning.

Epidemic management.—In epidemic management following points should be considered:—

(1) Detection of case.
(2) Hospitalization of patients.
(3) Segregation of contacts. Contact camps.
(4) Control of traffic and cordon.

Discussion

According to the author's experience pneumonic plague is found more frequently in the Western districts of the United Provinces. Pneumonic plague is not unknown in the eastern districts. I would suggest that the mask be made known to the public at large.

References

1. Gaud.—Sur l'importance du bubon sous-pectoral dans le diagnoistic et l'involution de la peste. *Bull. Off. d'Hyg. pub. inter.*, 1933.
2. Choksy.—A new type of plague.—Transactions of F.E.A.T.M. Calcutta, 1927.
3. Benzsonova.—Broth clouding varieties of B. pestis. *Rev. Microbio.,Epid.et Paras.* No. 3, 1929. Summarised in *Trop. Dis. Bull.* 1930.
4. Michelletti.—Examination of decomposing rats. *An. di. Med.*, Nov., Dec., 1932. Summarised in *Trop. Dis. Bull.* 1933.
5. Fusca.—Notions pratiques sur le diagnostic de la peste murine. *Bull. Off. d'Hyg. pub. inter.*, 1932.
6. Reports of Indian Plague Commission.
7. Greval and Dalal.—Agglutination of plague baccilli with high titre serum. *I. J. M. R.* October, 1933.
8. Flu.—Immunisition of rats against plague by means of Virulent plague bacteria. *Jl. Trop. Med. & Hyg.* Dec. 29.
9. Mention made in memoir on anti-plague vaccine in Indian Research Memoirs 1933.
10. Buchanan.—La preparation du vaccin anti-pesteuse a l'institut Lister. *Bull. Off. d'Hyg. pub. inter.*, pages 1626-30 October. 1932.
11. Taylor.—Memoir on anti-plague vaccine in India Research Memoirs 1933.
12. Naidu and Jung.—Standardisation of anti-plague vaccine. Transactions if F. E. A. T. M. Calcutta, 1927.
13. Sokhey.-Nouveau procede biologique de determination du pouvoir immunisant du vaccine anti-pesteux. *Bull. Off. d'Hyg. pub. inter.*, 1932.
14. Naidu and Jung.-Haffkine's plague prophylactic compared. *I. J. M. R.*, 1929.
15. Kapadia and Stevenson.—*I. J. M. R.* 1924-1925., also referred to in Enquete sur la vaccination anti-pesteuse in *Bull. Off. d'Hyg. pub. inter.*, 2932.
16. Referred to in Taylor's memoir on anti-plague vaccine in Indian Research Memoirs 1933.
17. Advier.—Etude d'un bacteriophage antipesteux. *Bull. Soc. Path. Exo.*, 1933. Summarised in *Trop. Dis. Bull.*, 1933.
18. Pais.—Influence des injections repetes de bacteriophage specifique sur l'involution de infection pesteuse experimentale du cobaye. *C. R. Soc. Bio.*, 1933 Summarised in *Trop. Dis. Bull.* Jan., 1934.
19. Reports Indian Plague Commission.
20. Novokovi and Lalazorv.—Transmission of plague through bed bug. *Rev. micro. epid. at parsit.*, 1931.
21. Referred to in Researches in parasitology of plague by Hirst.
22. Yamada.—Transmission of plague through the mite Liponyssus novogovyii. Transactions of F. E. A. T. M. 1930, Bangkok.
23. Patton.—*Jl. of Hygiene* November, 1924.
24. Hirst.—Enquete sur les puces de rats de Ceylon et breve discussion des recent travaux sur les rapports entre la distribution des especes de puse de rats et la propogation de la peste bubonique dans les Indes orientales. Reprint from *Ceylon Jl. of Science*,

February, 1933. Summarised in *Bull. off d'Hyg. pub. inter.*, January, 1934.

25. STRICKLAND AND ROY.—Calcutta Rat Flea Survey. *Trans. Roy. Soc. Trop. Med. and Hyg.*, March, 1930.
26. KING, IYER AND OTHERS.—Rat Flea Survey of Madras Presidency *I. J. M. R.*, 1929.
27. KING, IYER AND OTHERS.—Seasonal prevalence of rats and rat fleas,*I. J. M. R.*, April, 1933.
28. PANDIT, GEORGE AND OTHERS.—Rat Flea Survey of Ootacamund *I. J. M. R.*, 1933.
29. WEBSTER.—Anatomy of Xenopsylla larvae. *I. J. M. R.*, 1929.
30. HIRST.—Researches on parasitology of plague.
31. DE LA RUE.—Limportance de la puce de l'homme. *Bull. Soc. Path. Exot.*, 1932.
32. HIRST.—Researches on parasitology of plague.
33. Referred to in Problems of plague by F. P. Mackie. Trans. of F.E.A.T.M., 1927, Calcutta.
34 *Ibid.*
35. DORENBIS.—Underzuckungen over patho-genesis of plague der pest. *Nederl. Tej. Desh. v. Geness.*, February, 1930. Summarised in *Trop. Dis. Bull.*, 1930.
36. WILLIAMS AND KREMMER.—U. S. P. H. S. reports No. 33 August 17, 1923.
37 & 38. VARS.—Considerations sur l'anatomie pathologique de lo peste des rats sur. *Boll. offic. sanataria paramericana* Summarised in *Bull. Off. d'Hyg. pub. inter.*, January, 1934.
39. TEH.—Perpetuation of plague in rodents. Trans. of F. E. A. T. M., 1927, Calcutta.
40. JAMONSKY AND POLLISK.—On preserving plague virus in the organism of the fleas in suslike nests in non-epizootic period. Summarised in *Trop. Dis. Bull.*, 1933.
41. KUNHARDT AND CHITRE.—*I. J. M. R.*, January, 1921.
42. STRICKLAND.—Plague involving 2520 infected villages in Belgaum and Dharwar districts, *I. J. M. R.*, July, 1934.
43. GEORGE AND WEBSTER.—*I. J. M. R.*, July, 1934.
44. PANDIT.—Rat flea sunning. *I. J. M. R.*, April, 1933.
45. De l'influence de la sulphuration sur. le riz., *Bull. Off. d'Hyg. pub. inter.*, acut 1933.
46. NAIDU.—Treatment of plague. Transac. of F. E. A. T. M., 1927, Calcutta.
47. RAO.—Three cases of plague treated with Bayer 205. *I. M. G.* page 676, November, 1932.
48. System of Bateriology—Published by British Medical Research Council.
49. *Ibid.*
50. BENZSONOVA.—Hungry acid agar as differentiating medium for B. pestis and B. pseudo-tuberculosis rodentium. Summarised in *Trop. Dis. Bull.*, 1930.
51. KORRHKOVI.—Changement de pH et phenomene de reduction observe au cours de development de B. pestis and B. psuedo-tuberculosis rodentium *Ibid.*
52. WEBSTER.—Notes on plague *I. M. G.*, pages 214-216 April, 1933.
53. TEH.—Pneumonic Plague. Published by League of Nations.
54. THIROUX.—Resurches sur les causes de l'existence de la peste pulmonaire dans les regions froid et son absence dans leszones a temperature eleve aMadgascar. *Bull. Coc. Path Exot.*, October, 1929. (*Trop. Dis. Bull.* 1930).
55. BOYE.—Les relations entre la peste pulmonaire et la temperature. *Bull. Off. d'Hyg. pub. inter.*, Feb., 1930.
56. NIKANOROFF.—Plague in Russia. Trans. of F. E. A T. M. 27 Calcutta.
57. TEH.—Problems of Pneumonic Plague. *Ibid.*
58. NICOLLE AND OTHERS.—Vaccination preventive contre la pneumonie pesteuse par voie resperatoire. *Jl. Acad. Science.*, January, 1930.
59. SCUT.—Weiter Versuche mit Omnodin Bei Lungenpest in dem Tangier (Java) *Archiv. f. Schiff u. Trop. Hyg.*, April, 1930.

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EDITORIAL

AMOEBIASIS

Amoebiasis is an old standing endemic disease common in India and as more work is being done, more we are realising its far reaching consequences. Many cases of progressive ill-health with anaemia, torpid liver, acidity, hunger pain with periodic melaena, relapsing diarrhoea or colitis alternating with periods of chronic intestinal stasis and a few cases of stubborn cystitis have been found to be amoebic. Some cases of recurrent urticaria and subacute non-suppurative arthritis have been found to be due to escape of toxic amines or bacteria from the intestinal canal into the general circulation.

We draw the attention of our readers to the important issues raised by Dr. Majumdar in his paper on the subject published in the current issue pointing out the various shortcomings yet to be bridged over especially in the treatment of chronic amoebiasis. The value of an intensive course of treatment at the very beginning has been emphasised before much thickening takes place in the colon and that the treatment requires to be repeated periodically to ensure sufficient sterilization. The author has discussed the various drugs under two heads, *viz.*

(i) Those first absorbed into the general circulation and afterwards excreted at the site of the disease, *viz.*, colon or the liver.

(ii) Those acting more or less locally on the entamoebae in the intestinal canal.

In the first group, the important drug is emetine given by injection. If, however, much cicatrization has already formed and emetine cannot concentrate effectively there, as in cases of chronic intestinal amoebiasis with many relapses, the drugs of the second group are usually preferred. Kurchi alkaloids given by injection are readily excreted by the kidneys and do not concentrate effectively at the site of the disease.

The drugs of the second group include insoluble preparations of emetine for oral administration, kurchi alkaloids, bismuth salts, arsenical preparations as Stovarsol and Carbarsone, Yatren and Rivanol. It is obvious that a drug to be suitable for general use should be (i) of low toxicity, and (ii) cheap; and in this respect kurchi alkaloids are perhaps the best.

Of the arsenical preparations, Carbarsone has been found to be the best and is suitable for both acute (with emetine hypodermically) and chronic infections. It should not, however, be continued uninterruptedly for more than a week or 10 days

Inspite of considerable therapeutic advances, the problem of eradicating chronic intestinal amoebiasis is yet unsolved and this is because of the gross fibrotic changes in the gut wall which form fortified nests for the entamoebae and most of the amoebaecidal drugs fail to penetrate these.

CASE NOTES

GASTRIC DISCONNECTION (EISELBERG'S) OPERATION WITH GASTRO-JEJUNOSTOMY

LT.-COL. T. S. SHASTRY, I.M.S.

Sundaralingam Pillai, Hindu, Male, aged 46, admitted into the Government Headquarters Hospital, Palamcottah, on 5th February, 1936.

Complaint: Pain in the stomach since two years, coming between 1 a.m. and 6 a.m. daily with rumbling noises in the abdomen. Pain more felt on the right side, rarely on the left; vomiting of undigested matter between 3 and 4 p.m. about 3 hours after meals. Sour belching soon after meals.

General condition very poor, anaemic with pinched features.

After admission the stomach was washed regularly for four days; He was given anti-hook worm treatment too. Afterwards he was put on light nourishing diet for about a month. His pain and colour improved much.

Anaesthesia: 1. Local—½% Novocaine—preceded by morphine and atrophine. 2. Splanchnic—40 c.c. of ½% Novocaine.

Tip of gall-bladder adherent to a closely stenosed pyloric ring, proximal to which there was a uniform thickening of pyloric antrum; stomach was much dilated. The pyloric antrum was disconnected, the distal stump was closed, and a flap of gastro-hepatic omentum was stitched over it. The upper half of the stomach section was closed; the lower half was anastomosed to the jejunum. Abdomen was closed. Adrenalin ½ c.c. was given during the operation.

There was slight rise of temperature for 3 days after operation—maximum being 101°. After that the recovery was uninterrupted, sutures being removed on the 9th day of operation.

For the first two days after operation only glucose water was allowed. Then barley water with milk and buttermilk were given for about a week. Then he was put on good nourishing diet such as eggs, soup, liver, etc.

Simple gastro-jejunostomy provides, no doubt, a freer and more direct passage into the jejunum but it does not close the pyloric passage and so the irritation of the duodenal ulcer by the acid contents of the stomach and by the rough food particles is not stopped. Hence 'rest', mechanical and chemical, which is so much needed to allow the ulcer to heal is not secured.

Eiselberg's disconnection operation is thus a definite improvement on simple gastro-jejunostomy.

CHRONIC DUODENAL ULCER WITH GALL-STONES

Polya's Operation: Complication of Internal Strangulation

LT.-COL. T. S. SHASTRY, I.M.S.

A Hindu woman, aged 40 years, was admitted into the Women and Children Hospital, Vannarpet, on 11-3-36 at 5 p.m. for acute pain in the abdomen. Duration—about 4 months; but her history was not reliable as her behaviour was rather funny. She complained that she got acute pain 2 or 3 hours after meals, or even after taking any fluid, and that it was of a shooting character lasting for 3 or 4 hours, but relieved by taking any solids. No vomiting, but relief came after forced vomiting. She also got pain over the front and back of the chest on the right side, shooting up to the right shoulder.

General Health: Very emaciated. No. enlargement of spleen or liver.

Anaesthesia: (1) Local ½% Novocaine preceded by morphine and atropine. (2) Splanchnic with 40 c.c. of ½% Novocaine solution; a little $CHCl_3$ had to be given for closing the abdomen.

Stomach was dilated, pyloric ring was stenosed, a nodular hard mass of growth involving the first and second portion of duodenum was found; and this was found difficult to excise. The gall-bladder was distended, and contained two large, hard, sharp-pointed stones. The pyloric antrum was removed. The upper half of gastric section was closed, the lower half was anastomosed to the jejunum through the gastro-colic omentum (Polya's operation).

For the first two days only glucose saline and sodium bicarb. drink—then diluted milk. Put on rice and *Rasam* (pepper water) on the fifth day. No temperature. Pulse good throughout. Wound clean. On the sixth day there was a little superficial pus in the skin sutures—so a few of the latter were removed and drainage established—afterwards recovery was uninterrupted.

On 28th March 1936, that is, a fortnight after the operation, she complained of colicky pain again. This must be due to the gall-stones. So she was kept for a second operation. But she absconded on 13th April, 1936. She returned 3 days after, *i.e.*, on 16th April, 1936, in a collapsed state, cold and clammy, pulseless, etc., with severe colicky pain and vomiting of colourless fluid—no motion for 3 days. The abdomen was soft; but a slight distension was seen on the left side of the abdomen, which slowly began to increase in size. She vomited greenish fluid. The stomach was washed, and also Pituitrin injection 1 c.c., given; and she was taken to the Theatre after two pints of hypertonic saline was given intravenously and afterwards the general condition and pulse improved.

16th April, 1936.—Abdomen was opened in the midline and under local anaesthesia preceded by morphine and atropine injections. Some adhesions were found; after these were cleared a coil of jejunum was found, having passed through the osteum in the transverse mesocolon by the side of the gastro-jejunal anastomosis. This coil was pulled back easily and the osteum was closed, and the operation completed.

The solid mass found round the doudenum at the first operation (12-3-36) and thought to be of a malignant character was now found reduced to about one-third of its original size. Most of it must have been absorbed—it evidently was a chronic inflammatory mass due to the chronic ulcer.

Recovery un-interrupted. Discharged cured on 24th May, 1936, wellnourished and normal.

Simple gastro-jejunostomy has been found to give rise to secondary gastro-jejunal ulcer, *i.e.*, ulceration at the anastomosis, the treatment of which is more difficult and sometimes impossible. Hence the Vienna School following Billroth has evolved partial gastrectomy, and the results over a large number of years have been found to be very satisfactory. This operation is more severe, no doubt, than the old operation of simple gastro-jejunostomy. But the convalesence after partial gastrectomy is much easier and happier for all—patient, nurse, and surgeon.

The shock can be further reduced if chloroform could be avoided. Here again Finsterer's method of (1) local anaesthesia for the abdominal wall, and (2) splanchnic anaesthesia for internal work has been found to be excellent.

Special Features

(1) She sought admission and pressed me to operate because of the severe pain in the epigastrium due to the duodenal spasm and gall-stones.

(2) The hard nodular large mass in the duodenum looked and felt like carcinomatous at the first operation.

(3) The complication of strangulation.

(4) The duodenal mass has become reduced in size and hardness in 35 days—so, it must have been due to hyperplastic granulomatous process at the site of ulceration, but has got reduced remarkably, because by partial gastrectomy the passage of irritating food and acid contents and the consequent spasm, were abolished, absolute rest was secured.

(5) The presence of gall-stones and cholecystitis must be noted. It is possible that the duodenal ulceration gave the incentive to inflammation and stone formation in the gall-bladder.

(6) The happy result of the second operation, as it was done before the stage of actual toxaemia developed.

MODERN THERAPY IN GENERAL PRACTICE

DIFFICULTIES IN THE TREATMENT OF AMOEBIASIS

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The grosser manifestations of amoebiasis, especially of the intestine, are often so easily controlled, that one is apt to think that the treatment of the disease leading to cure is a straightforward affair. The more we realise how vague and indefinite the symptoms of the disease may be and how often after the disappearance of the active symptoms some residual infection may persist for months or years and even life-long, causing much distress and sometimes permanent invalidism to the sufferer and risk of spread of the infection to the contacts, we feel, inspite of much advance, that something more is yet left to cover up.

The most important drug for the treatment of amoebiasis is undoubtedly the preparations of Ipecacuanha.

Ipecacuanha: The dried roots were introduced to Europe from Brazil by PiSo in 1648[1] and was given by him in chronic dysentery in the form of infusion in drachm doses. This was soon replaced by powdered root given in 20 to 30 gr. doses at bed-time at first with Calomel or Blue pill (Twining) and afterwards more often with opium. A relic of that is yet left in Dover's powder (Macgregor) though no longer used for the treatment of amoebiasis. Its usefulness both for intestinal and hepatic amoebiasis [2,3] was well established.

The alkaloids of ipecac., then collectively called emetine, were isolated by Pelletier (1817) and were probably given orally by Bardsley (1829)[4] for chronic diarrhoea and dysentery.

But the main difficulty was nausea and vomiting which frequently followed its administration even with a preliminary dose of opium or chloral and bromide. Simon and Dock (1909) tried salol-coated Ipecac. pills 5 gr. each, 10 to 15 gr. being given at bed-time. This was an improvement but did not solve the problem. So some authorities advised sine-emetine ipecac., a misdirected ingenuity.

Luckily in 1911, the things took a happy turn. Vedder [5,6] published his important experiments showing that emetine had the most powerful action on E. histolytica and that it was capable of being absorbed into the circulation to act on the entamoebae in the tissues of the large intestine and the liver.

Rogers[7] quickly followed him and in 1912 he published his reports showing that emetine could be safely injected subcutaneously without causing nausea or vomiting and obtained remarkably rapid cure of a large number of acute and chronic cases of intestinal amoebiasis. The parasites disappeared from the stool rapidly. Equally favourable results followed in amoebic hepatitis and hepatic abscess. He further showed[8] in 1914 that the other alkaloid of ipecac., cephaeline was much less effective than emetine and a mixture of emetine and cephaeline in proportion as they exist in ipecac., was intermediate in action. Rogers deserved the credit for being the first to fix up the right method of the treatment.

The results were so readily confirmed by other workers too numerous to mention, that injection of emetine is now the standard treatment for acute amoebiasis.

The immediate results were so satisfactory that Rogers thought that by giving hypodermically or intramuscularly two 1/2 gr. doses or one 1 gr. dose daily for a few days, all the amoebae in the body could be completely killed. But its deficiencies were soon realised.

The problems that next presented were to find out,

(1) What could be the safe daily dose of emetine and for how long it should be continued?

(2) Was complete sterilization, as hoped for, achieved by emetine and if not, what else could be done to have it?

(3) Could the painful injections be dispensed with by providing for an effective substitute, suitable for oral administration?

1. *The optimum dose of Emetine*:

Emetine given by injection acts by being excreted into the colon to come in close contact with the entamoebae embedded in the tissues (Lyons 1915)[9]. Locally, it is highly irritant to the intestinal mucous membrane. It is even more toxic to the cardiovascular and the nervous systems. Further emetine is cumulative (Walter and Koch, 1917)[10].

Consequently various toxic phenomena may follow administration of too big a dose (especially if intravenous) or repetition of injections too long without intervals. These are:

(i) Frequently relapsing and obstinate diarrhoea or colitis.

(ii) Cardio-vascular depression (Myocarditis) and periodic attacks of palpitation.

(iii) Nervous depression leading to inability for prolonged muscular and mental exertion.

(iv) Sometimes peripheral neuritis, causing wrist, ankle and toe drop (Lyons, 1915, Johnson and Murphy[11], Kilgore[12], 1917).

Pregnant and menstruating women should be given a smaller dose (Chalmers and Papatheodoru[13], 1916).

The dose we used with safety for an adult male with acute amoebiasis is one gr. hypodermically daily for 6 to 9 days followed by an interval of 3 to 7 days and then another 3 injections. If for any reason it was thought necessary to continue the treatment, more courses of 3 injections were given at weekly intervals. Though a subcutaneous injection sometimes causes superficial ecchymosis, it is less painful than intramuscular injection. Pain may be lessened by making an isotonic solution of emetine with sodium chloride.

Intravenous injection is not necessary unless the case is so severe as to threaten gangrene of the intestine. I had to give several times in such severe cases $\frac{1}{2}$ gr. with 5 c.c. of 20% glucose solution very slowly in the morning and $\frac{1}{2}$ to $\frac{3}{4}$ gr. subcutaneously in the evening with rapid improvement in cases not readily responding to subcutaneous injections[14]. I had no alarming symptoms. Rogers also advocated such a measure.

In pregnant women, $\frac{1}{2}$ to $\frac{3}{4}$ gr. should be the usual dose. In one such case with acute hepatitis, where one $\frac{1}{2}$ gr. and one 1 gr. emetine injection hypodermically did not show sufficiently rapid response, $\frac{1}{2}$ gr. was given intravenously on the third day. The result was very good and this caused no harmful effect on the uterus.

2. *Is complete sterilization possible with Emetine?*

Emetine like other drugs having chemo-therapic action on the protozoa, failed to satisfy Ehrlich's hope of effecting complete sterilization by a single massive dose. In nearly every acute case, 2 or 3 injections of emetine cause a remarkable change: the pain and tenderness in the abdomen disappear and the stool becomes nearly normal, the parasites disappearing.

But this clinical cure is not associated with complete disappearance of the parasites from the tissues. So unless a well-regulated course of treatment is persued, a fair number of cases relapses: there being relapsing diarrhoea or colitis, sluggish liver or actual hepatitis, dyspepsia, progressive anaemia or other more obscure manifestations.

These difficulties were realised soon after the introduction of emetine. Several factors have been thought to be responsible. These are:

(i) Insufficient courses of emetine, not enough being given at the acute stage; or probably emetine, like arsenobenzols in the treatment of syphilis, can only rapidly bring down the parasitic level to a minimum and other remedies have to be sought for, for final sterilization.

(ii) Co-existing or superimposed secondary infections as by various dysentery bacilli, haemolytic streptococci or other intestinal organisms which either impair the vitality of the invading mucous membrane or lessen the activity of the amoebicidal drug.

(iii) On account of long standing ulcerative processes in the intestine so much scarring sometimes take place that any drug given by injection does not reach the site of the infection sufficiently readily and the entamoebae have made for them-

selves in the scar tissue fortified nests where they live safely and consequently resist treatment. Drugs by oral administration may be better in such cases.

So other preparations of ipecacuanha orally and also other amoebicidal drugs had to be searched for. Many such have been introduced from time to time and more yet are coming in but the problem is not completely solved. We discuss the action of the important drugs of the series.

(i) Keratin coated emetine pills orally:

Low[15] (1913) found these useful in mild cases of amoebiasis. I used these in Rogers's Ward (1913) in the Calcutta Medical College Hospital. But the results were not very satisfactory. The pills were in many cases vomited out as probably the coating got cracked and some emetine escaped into the stomach. These are no longer used in practice.

(ii) Jones[16] (1915) advised a combination of large doses of ipecacuanha by the mouth with hypodermic injection of emetine which he thought would have more intensive effect but the actual results were no better.

Mac Adam[17] (1919) gave 1 gr. hypodermically and ½ gr. by the mouth at bed time for 12 days. The results were not very encouraging and the dose was not too safe.

(iii) Stephens and Mackinon[18] (1917) reported on *Alcestra Ipecac*. This consists of alkaloids of Ipecac. adsorbed with hydrated aluminium silicate made into 5 gr. tablets, and 5 tablets are given twice daily containing in all 1½ gr. of emetine. These pass through the stomach unchanged and so do not cause much nausea and vomiting. These had some popularity and are sometimes given in chronic relapsing cases even upto the present day.

(iv) Various insoluble emetine preparations have been introduced: These pass through the stomach more or less unchanged but liberate emetine in the alkaline secretions in the small intestine.

DuMez[19] (1915) prepared *Emetine Mercuric Iodide* and *Emetine Bismuth Iodide*. The latter attained much popularity and proved to be of great promise for several years. Dale[20] (1916), Low and Dobell[21] (1916), Imrie and Roche[22] (1917), Dobell and Low[23] (1916), Low (1917), Waddell, Banks, Atson and King[24] (1917) and Jepps and Meakins[25] (1917) were among the earlier observers to report favourably on it in chronic relapsing amoebiasis and for the carrier state. It is given in 1 gr. cachets 3 times daily (Dobell, 1916), in 2 gr. pills (Lambert, 1918)[26] or as an emulsion containing 3 gr. in ½ oz. of liquid paraffin taken in 2 or 3 oz. of water (Jepps, 1921)[27]. Altogether 36 gr. are taken in one course which might be repeated after one week. Salol or Keratin coated pills of E.B.I. do not dissolve readily and so should not be given.

Usually, it is given in cachets or in paraffin emulsion at bed-time which is preceded by a dose of opium. When the patient is nearly sleepy, the drug is given.

It contains about 30% of emetine as its constituent; it is probably emetine that counts and not so much iodine and the daily dose contains a little over 1 gr. of emetine. It has also been given along with emetine hypodermically (Watson-Wemyss and Bentham, 1918)[28] but more commonly during the intervals of emetine injections.

Disadvantages are (i) nausea and vomiting which are so marked in many cases as to cause more disagreeable symptoms than emetine hydrochloride by the needle (Low and Dobell, 1916)[23]. In a favourable case, however, a certain amount of tolerance is soon established.

(ii) The liberated emetine causes local irritation on the intestine, and if continued long, may cause emetine diarrhoea or colitis. It is consequently unsuitable in acute amoebiasis: in such cases, it may cause severe haemorrhage (Manson-Bahr, 1921)[29]. Doubtless many patients did not like it and I had difficulties also. Yet it has a distinct place in the treatment of relapsing chronic amoebiasis and carrier state.

Other insoluble preparation is *Emetine Periodide* in 2 gr. pills given with 5 gr. of desiccated ox bile, 3 times daily for 5 days (Willmore, 1923)[30]. This contains about 38·7% of emetine. Low, Castellani, Weynon and Manson-Bahr had some favourable results.

Auremetine: A combination of auramine, an aniline dye, and emetine, containing 28% of emetine, is given in 1 gr. doses in gelatin capsules 4 times daily for 10 to 15 days. On alternate days Stovarsol is also given 2 or 3 times daily. It is believed to be more effective than E.B.I. and does

not cause nausea, vomiting and purging (Willmore and Martindale, 1926)[31].

But these have not attained the popularity of E.B.I.

Other Drugs used for the Treatment of Amoebiasis

(i) *Bismuth subnitrate*:

This is being used by Deeks[32] in Panama since 1908, 180 gr. suspended in a tumbler of water taken every 3 hours, night and day. Deeks and Shaw[33] (1911) reported that this method of treatment was very successful and they never used Ipecac. and declared that they hardly had any relapse. Amoebae disappeared from the stool readily. In 1914, Deeks combined bismuth orally with emetine hypodermically and Deeks and James[34] (1925) showed that this combined treatment was very successful in preventing relapses. Bates[35] (1916) also found large doses of bismuth useful after a course of ipecac. or emetine. Acton and Knowles[36] (1924) advised bismuth carbonate in 2 dr. doses every 4 hours, emetine being given 2½ hours after the first dose. They thought this would raise the alkalinity of the portal blood stream in which condition the suggestion was that emetine would act better.

We also prefer bismuth carbonate but for a different reason; subnitrate is decomposed in the intestine to form bismuth sulphide and nitrous oxide and the latter when such big doses are being given may cause nitrite poisoning.

We give bismuth carbonate in 1 dr. dose with standardised liquid extract of Kurchi 3 or 4 times daily in all acute and subacute cases. The local action of bismuth and of Kurchi tannin and gum resins on the ulcerated surface forming a mildly astringent protective coating is undoubtedly helpful.

(ii) *Kurchi alkaloids*:

The concentrated decoction of the bark is a reputed medicine in the treatment of chronic diarrhoea and dysentery in Ayurveda. The alkaloids collectively are called Kurchi alkaloids and these have been found to be active amoebicide (Brown[37], 1922 and Chopra, Gupta, David and Ghosh[38], 1927) and in experimental condition, nearly as active as emetine.

The first preparation containing a sufficient amount of the alkaloids was prepared by Brown (1922) in B. W. & Co.'s, laboratory in the form of tablets and favourably reported on (Acton and Knowles,[39], 1928). But these are unthinkably expensive. The first standardised preparation convenient for general use with 1% of the alkaloids was prepared at my instance by Messrs. Bengal Chemical & Pharmaceutical Works (1928)[40]. This also contains 0·25% of tannin and some gum resins and being cheap and also effective is now largely used in practice. An insoluble preparation apparently after E.B.I. of DuMez has also been prepared (Acton and Chopra[41], 1929). We have made an extensive clinical investigation of the therapeutic possibilities of Kurchi alkaloids and our considered opinion is as follows: (a) Kurchi alkaloids, compared with emetine are slower in action and less effective but being non-toxic and not nauseating can be given orally in 3 to 4 gr. doses (6 to 8 fl. dr. of the standardised extract) and for a prolonged period (Majumdar, 1930)[42].

The convenient way in an acute case is to combine emetine injections hypodermically with liquid extract of Kurchi 2 dr. with 1 dr. of colloidal kaolin or bismuth carbonate made up with chloroform water, 3 or 4 times daily. The Kurchi mixture is continued even during the intervals of emetine injection and in this way may be given uninterruptedly for about one month or more. Such a combined treatment is more effective than either of these separately. (b) Tablets of the solid extract or of Kurchi bismuth iodide have no advantage except the convenience of transport. In an acute case these are often passed out entire in the stool (Majumdar, 1930)[43] and I have recovered them entire from the intestine at the P.M. examination. In a chronic case, we prefer tablets of the solid extract like one prepared by B.W. & Co. or Kurchidin (Bose's Laboratory) rather than Kurchi bismuth iodide as unlike emetine, Kurchi alkaloids not being emetic, the need for an insoluble compound does not exist and such a compound has the disadvantage of slow diffusability. (c) Injections of Kurchi alkaloids, hypodermic, intramuscular or intravenous, even in 2 or 3 gr. doses daily have not given us as dependable a result as even ½ gr. of emetine (Majumdar, 1933)[45]. This is because as we found the alkaloids are readily excreted in the urine and do not effectively concentrate on the colon at the site of the disease.

(b) Tannin of Kurchi has no special amoebicidal effect (Majumdar, 1931)[46] but this with gum resin and bismuth carbonate in a fairly big dose, prescribed in the form of a mixture, form a protective coating which favours better healing of the ulcers.

(iii) *Yatren* 105:

This is a complex organic compound containing 26 to 28% of iodine introduced by Muhlens and Menk (1921). This is given in 4 gr. keratin-coated pills, one 3 times daily and sometimes in the form of enema also, 200 c.c. of 2·5 to 5% solution being given for about 10 days: this is retained by the patient as long as possible.

Menk[46] (1922) considered it useful in chronic amoebiasis and advised its use as enema for 5 to 6 weeks and to repeat it at 6 monthly intervals. Manson-Bahr and Morris[47] (1925) also found it useful, orally 2 pills twice daily or enema of 2·5 to 6% solution for 8 to 14 days: in resistant cases, a combination of the two: Craig[48] (1934) considers it to be a very good amoebicide. He apparently wants to put it on the top even above emetine. Our experience with this drug is limited. Its price is so high that it is not likely to be a drug of choice. It should rather be given a chance in resistant cases in the intervals of other amoebicides. It is non-toxic and this is its attraction.

(iv) *Rivanol*:

This is an acridine dye given orally in capsules and also in 1 in 1000 sol. as enema and is reported to be a good amoebicide (Urchs, 1927)[49]. Through the courtesy of Messrs. Havero Trading Co. we got a liberal supply and tested the capsules orally and also enema in the Campbell Hospital but the results were unsatisfactory (Majumdar, 1931)[50]. 6 capsules given daily for 2 days did not make the emoebae disappear from the stool and only two injections of emetine of ½ gr. each for the next 2 days made remarkable change. Biggam[51] (1930) also found similar unsatisfactory results. De-hydronol[52] and Vioform[53] are recently introduced and we have to wait for the result. Oil of chenopodium [54,55] is of not much outstanding value.

(v) *Arsenical Compounds*:

Salvarsan[56], 0·1 gr. in cachets 5 to 6 times daily orally rapidly reduced to 2 or 3 times daily (Ravaut[57], 1926). Neosalvarsan intravenously (Reed, 1922)[58] did not receive much support.

Spirocid or Stovarsol in 4 gr. tablets is more frequently given orally in chronic amoebiasis, 2 or 3 times daily for 7 days: an interval is given for 7 days and then one tablet daily for another 7 days. This has been introduced by Marchoux and favourably reported by Gorine and Lepine[59] (1924), Petzetokis[60] (1925) and Manson-Bahr[61] (1925). Craig[62] found it of value for the carrier state (1928).

Local representatives of the manufacturers very kindly put at my disposal sufficient quantities of each for clinical trial. These were of no value in acute amoebiasis, 3 tablets daily for 3 days, did not make the entamoebae disappear from the stool though they rapidly yielded to ½ gr. emetine daily for 2 days. These have, however, been found of value in weak and anaemic patients suffering from chronic amoebiasis acting more as a general toxic than a direct amoebicide and are best given in the intervals between the successive courses of emetine.

It must be remembered that the drug is fairly toxic and may cause alarming symptoms especially in susceptible persons. If continued long, it may cause gastro-enteritis, oedema of the eye-lids, skin eruptions and like other pentavalent aromatic arsenic compounds, optic neuritis.

Treparsol[63], an allied preparation, is also similarly used and is probably as good.

Carbarsone, first prepared by Ehrlich, has recently been introduced for the treatment of amoebiasis by Anderson and Reed[64] (1931). It is given in 4 gr. doses in gelatin capsules twice daily for 10 days and another course, 10 days after in resistant cases. They gave it per rectum also in refractory cases as enema of 200 cc. containing 1% of Carbarsone and 2% Sodi Bicarb., the drug being soluble in alkalies (1934)[65]. Other observers also favourably reported on it.

In a limited number of cases treated by me, I found that in acute cases, it is less active than emetine (a similar view is held by Calender[66], 1935) and in subacute and chronic cases it is effective but should preferably be given along with or alternating with emetine. It is of lower toxicity than Stovarsol but may sometimes cause optic neuritis.

CONCLUSION:

(1) It will therefore appear that the treatment of intestinal amoebiasis is comparatively easy so far as the relief of acute symptoms is concerned and the first drug of choice is emetine hypodermically.

(2) For subacute, chronic or relapsing cases and for the carrier state, many drugs have been proposed, mostly given orally but none have the record of uniform success in all cases.

(3) As in such cases the treatment has to be continued fairly long, a drug that combines efficiency with cheapness and low toxicity should have preference.

(4) On the analogy of treatment of syphilis with arsenobenzols alternating with bismuth or mercury, the treatment of amoebiasis should also be a combined one, rapidly acting emetine should be supplemented by drugs of slower and more sustained action. With the above object in view, the scheme that I followed with much success is as follows:

(i) In all cases of acute or subacute dysentery the patient should be kept in bed and given liquid or semi-solid diet.

(ii) A does of castor oil is given at the start to flush out the toxic debris lying in the intestine. No purgative is given afterwards as this takes away a part of the specific from the intestinal canal but locking of the bowels is prevented by liquid paraffin or any other mild laxative given at bed-time.

Mucilaginous drinks as of Bael (*Aegle marmelos*) and of Ispaghul (*Plantago Ovata*) are helpful.

(iii) In all acute cases emetine hydrochloride is given hypodermically (1 gr.) for 6 to 9 days followed by an interval of 7 days and in a severe case, 3 days and then 3 injections are given daily on every other day. Several such short courses may be necessary. In a chronic case, not more than 6 injections are given at the start and 3 injections at weekly intervals are given for several weeks.

(iv) Standardised liquid extract of Kurchi 2 dr. with 1 dr. of bismuth carbonate or colloidal kaolin flavoured with syrup and chloroform water should be given along with it, 3 or 4 times daily orally for one month and also periodically afterwards.

(v) In refractory cases, though their number will be comparatively few if the initial course of treatment is intensive, along with several short courses of emetine hypodermically, Emetine bismuth iodide alternating with Carbarsone should be given orally.

I am indebted to the Editors of Medical Annual and Tropical Diseases Bulletin for references.

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REFERENCES

1. MORHEAD's Clinical researches on Diseases of India (1860).
2. CHEVERS' Diseases of India (1886).
3. ROGERS, *Practitioner*, June, 1907.
4. CREIG, Amoebiasis (1934).
5. VEDDER, *Journ. Trop. Med. & Hyg.*, 1911, 149.
6. VEDDER, *Jour. Am. Med. Assoc.* 1914, 501.
7. ROGERS, *Br. Med. Jour.*, 1912, June 22, and Aug. 24.
8. ROGERS, *Ind. Med. Gaz.*, 1914, 85.
9. LYONS, *Am. Journ. of Trop.* Dis. 1915, 663.
10. WALTH AND KOCH, *Journ. Exp. Pharmacology*, July, 73.
11. JOHNSON AND MURPHY, *Military Surgeon*, 1917 March, 286.
12. KILGORE, *China Med. Jour.*, 1917 May, 121.
13. CHALMERS AND PAPATHEODORU, *Journ. of Trop. Med.* in *New York. Med. Journal*, 1916, Dec. 4.
14. MAJUMDAR, *Advance Therapy*, July 1931, 55.
15. LOW, *Br. Med. Jour.* i. 1369.
16. JONES, *Jour. of Am. Med. Assoc.*, 1915, i, 912.
17. MAC ADAM, *Ind. Jour. of Med. Research*, Jan., 1919.
18. STEPHENS AND MACKINNON, *Ann. Trop. Med. and Parasitology*, Feb. 1917, 347.
19. DUMEZ, *Philadel. Jour. of Sci.*, Sec. B., 1915, 73.
20. DALE, *Lancet*, 1916, ii, 183.
21. LOW AND DOBELL, *Br. Med. Jour.*, 1916, ii, 612.
22. IMRIE AND ROCHE, *Lancet*, 1917, i, 17.
23. DOBELL AND LOW, *Lancet* 1916 ii, 319.
24. WADDELL, BANKS, WATSON AND KING, *Lancet* 1917, ii, 73.
25. JEPPS AND MEAKINS, *Br. Med. Jour.*, 1917, ii, 645.
26. LAMBERT, *Br. Med. Jour.*, 1918, i, 116.
27. JEPPS, *Journal of R.A.M.C.* 1921, June 443.
28. WATSON-WEMYSES AND BENTHAM, *Lancet* 1418.
29. MANSON-BAHR, *Br. Med. Jour.*, 1921, ii, 115.
30. WILLMORE, *Royal Society of Trop. Medicine*, London, May, 1923.
31. WILLMORE AND MARTINDALE, *Br. Med. Jour.*, 1926, i, 525.
32. DEEK, *Medical Record*, November 13, 1909.
33. DEEK AND SHAW, *New Orleanz Med. and Surg. Journ.*, July, 1911.

34. Deek and James, *Am. Journ. of Trop. Med.* 1925, March, 97.
35. Bates, *Jour. Am. Med. Assoc.*, 1916, ii., 346.
36. Acton and Knowles, *Ind. Med. Gaz.*, July, 1924.
37. Brown, *Br. Med. Jour.*, 1922, i, 993.
38. Chopra, Gupta, David and Ghosh, *Ind. Med. Gaz.*, 1927, March, 132.
39. Acton and Knowles, On the Dysenteries of India (1928).
40. Majumdar, *Advance Therapy*, January, 1929.
41. Acton and Chopra, *Ind. Med. Gaz.*, September, 1929.
42. Majumdar, Modern Pharmocology and Therapeutic Guide, 2nd Ed. (1930).
43. Majumdar, *Ind. Med. Gaz.*, February, 1930.
44. Majumdar, Modern Pharmacology and Therapeutic Guide, 3rd Ed. (1932).
45. Majumdar, *Journ. Trop. Med. & Hyg.*, August 15, 1931.
46. Menk, *Munch. Med. Woch.*, 1922, September 1, 1280.
47. Manson-Bahr and Morris, *Lanct* 1925, ii, 544.
48. Craig, Amoebiasis (1934).
49. Urchs, Transaction of the F. E. A. T. M., 7th Congress, December, 1927.
50. Majumdar, *Advance Therapy*, July, 1931.
51. Biggam, *Lancet*, 1930, i, 1335.
52. Ratchiff, *Am. Journ. of Trop. Med.*, 1931, July.
53. Giordano, *Arch. Ital. Sci. Med.*, Colon., 1934, September. 1.
54. Walker and Emrich, *Jour. Am. Med. Assoc.*, 1917, ii, 1456.
55. Wadham and Cart, *Jour. Am. Med. Assoc.*, 1918, ii, 356.
56. Wadham and Hill, *Jour. Am. Med. Assoc.*, 1913, ii, 385.
57. Racart, *Presse Med.*, 1926, April 21.
58. Reed, *Am. Jour. of Med. Sci.*, 1922, October, 586
59. Garin and Lepine, *Presse Med.*, 1924, November, 22
60. Petzetokis, *Presse Med.*, 1925, March 7.
61. Manson-Bahr and Morris, *Lancet*, 1925, ii, 546
62. Craig, *Jour. Am. Med. Assoc.*, 1928, April, 28.
63. Vialard and Darleguy, *Presse Med.*, 1925, June 6
64. Anderson, Reed, David and Leoke, *Jour. Am. Med. Assoc.*, 1932, January 15.
65. Anderson and Reed, *Amer. Jour. Trop. Med.*, 1934, March.
66. Calender, *Amer. Jour. Trop. Med.*, 1935, March

CURRENT MEDICAL LITERATURE

MEDICINE

Thrombopenic Purpura

Fowler (*Annals of Int. Med.*, May, 1936, page 1475) reports on the study of analysis of 160 cases of thrombopenic purpura with regard to its etiology, prognosis and treatment. The author observes that of these 160 cases 143 are of the secondary type and that infection plays a great part in the causation of secondary thrombopenic purpura and also acts as a precipitating factor in the idiopathic cases. Proper treatment depends on accurate diagnosis and consists of special diets of high vitamin intake and calcium salts, antivenin, peptone shock therapy, roentgen-rays over the spleen and finally splenectomy. Splenectomy should be deferred till it has been determined whether the attacks are becoming more severe, and till the effects of transfusion have been noted and all sources of infection have been throughly eradicated.

The Therapeutic Effect of Solution of Potassium Arsenite in Chronic Myelogenous Leukaemia

Stephens and Lawrence (*Annals of Int. Med.*, May, 1936, page 1488) report on the effective palliative treatment of 7 cases of chronic myelogenous leukæmia with prolonged courses of solution of potassium arsenite administered in toxic or sub-toxic doses. The authors note that the drug when used in the above manner produces (a) symptomatic improvement, (b) reduction in the size of the spleen, (c) decrease in total white blood cell count, (d) decrease in the number of immature cells and in the number of nucleated red cells in the peripheral blood, (e) improvement in the anaemia, (f) increase in platelets. Though symptomatic and pathologic relapses occur during the course of treatment with the solution of potassium arsenite this treatment may be tried with advantage in chronic myelogenous

leukæmia in conjunction with or alternating with treatment with roentgen ray.

Control of Addison's Disease with a Diet Restricted in Potassium

WILDER AND HIS ASSOCIATES (*Proc. Staff. Meet. Mayo Clinic*, 1936, xi, page 273-83) in reporting the results of their investigation in the Mayo Clinic on the control of Addision's disease observe that if the potassium intake of a patient is restricted to 1·6 gm. per day the sodium and chloride intake can be reduced from such figures as 8·1 gm. sodium and 10·9 gm. chloride to 6 gm. of sodium chloride without altering the percentage of sodium and potassium in the blood and without producing crisis and that if the potassium intake is raised from 1·6 gm. to 4 gm. per day there is immediate loss of sodium and chloride in the urine with fall of blood sodium and development of crisis which again is to be tackled with cortin and intravenous salt. The authors conclude by observing that the successful treatment of the disease requires that the potassium content of the diet must not exceed 1·6 gm. a day.

Treatment of Pneumonia with Serum containing Type-Specific, Heterophile, and Neutralising Antibodies

CALLOMON AND UNGAR (*Annals of Int. Med.*, June, 1936, page 1664) in giving an analysis of 32 cases of pneumococcus pneumonia treated with serum corroborates the observations of other investigators that the efficacy of the type-specific anti-pneumococcus horse serum is enhanced by the addition of heterophile and neutralising antibodies in the pneumococcus filtrates. The authors observe that cases treated with such modified serum are found less toxic, their course become shorter and that this serum is particularly useful in Type III and Group IV cases which are not easily amenable to treatment with the conventional anti-pneumococcus serum. The authors conclude that this is certainly a distinct advance in the treatment of pneumococcus pneumonia.

Diabetes Refractory to Insulin, with Report of a Case

ALTSHULER (*Annals of Int. Med.*, May, 1936, page 1602) in reviewing the literature on the subject and reporting a case of diabetes which was entirely refractory to insulin observes that during the period she was under observation the hyperglycæmia and glycosuria had no relation to the amount of insulin injected and that ketosis only resulted each time when insulin was totally omitted. The author contends that this refractoriness is due not to any dearth of insulin but to some factor inhibiting or antagonising the action of insulin. The autopsy of the case showed a large suprasellar cystic haematoma pressing on the hypothalamic structure including the hypophysis, pointing to the pituitary as the source of the antagonistic substance and there was nothing both clinically and pathologically to show that the pituitary effect had come indirectly through these glands. The author concludes that the refractoriness to insulin in cases of glycosuria may be taken as a diagnostic sign of the presence of a lesion in the hypophysis.

Late Toxic Results including Dermatitis Exfoliativa, from Dinitrophenol

HITCH AND SCHWARTZ (*Journ. Amer. Med. Asson.*, June 20, 1936, page 2130) report a case which developed exfoliative dermatitie, cataracts and polyneuritis after taking only 28 grams of dinitrophenol over a period of 14 days. The most striking manifestation was the violent cutaneous reaction which the patient had and it was exactly like that following arsphenamine therapy.

Glucose in Hyperpiesis and Acute Pulmonary Oedema

POLACK AND HARPOTH (*Hospitalstidende*, Feb. 11, 1936, page 141) report on the successful treatment of various forms of high blood pressure and also in acute pulmonary oedema with intravenous injection of hypertonic glucose solution. The authors write that the blood pressure fell considerably in all but three cases and there was prompt relief of symptoms such as headache of uraemia. They observe that the injections are quite safe and very efficacious in reducing blood pressure and may be a useful supplement or alternative to other measures that are usually followed. The authors further observe that the therapy is highly indicated in acute oedema of the lungs when 50 c.c. of a 50% glucose solution is put into a vein after withdrawing some blood from it and the result is often marvellous.

Further Observations on the Gold Treatment of Rheumatoid Arthritis

HARTFALL AND GARLAND (*Lancet*, June 27, 1936, page 1456) report on the successful treatment of rheumatoid arthritis with gold. The authors treated 374 cases with cure or marked improvement in 78% and slight improvement in another 15%. They observe that the single dose must not exceed 0.1 g. and the total amount of gold in each course must not be more than 1.0 g. and that each patient should get at least two courses of injection with an interval of not less than 3 months' duration. The drug may be used intravenously or intramuscularly as this has made in the series no marked difference in the curative effects nor in its toxicity.

SURGERY

Acute Staphylococcus Osteomyelitis

JOYNER AND SMITH (*Surg. Gynec. Obstet.*, July, 1936, page 1) report that 16 patients with acute osteomyelitis and staphylococcæmia when treated with prompt drainage and repeated blood transfusion resulted in eight deaths and eight recoveries. 13 patients with same type of infection had similar treatment with the addition of staphylo- had similar treatment with the addition of staphylococcus antitoxin and the result was 2 deaths and 11 recoveries. The authors observe that the Schilling index is an indicator of the severity of the toxæmia and a convenient guide to the dose of antitoxin and frequency of its administration.

Prevention of Recurrent Renal Calculi

HIGGINS (*Surg. Gynec. Obstet.*, July, 1936) observes that the usual routine treatment does not reduce the percentage of recurrence below 10 to 15%. He further observes that a group of 31 patients who had renal colic before and who passed stones spontaneously have been free from symptoms for more than 2 years when they were put on strict dietetic management following operation *viz.*, a high vitamin A, alkali or acid ash diet, the contituents of which depended on the hydrogen-ion concentration of the urine and the chemical constituents of the calculi. The recurrence percentage is now 4·7 and this includes some cases who underwent no x-ray examination immediately after operation and who might have got some stones still left.

Congo Red for Control of Bleeding

GRAVES AND KICKHAM (*New Eng. Journ. Med.*, April 16, 1936, page 782) report on the success of congo red as a hæmostatis agent in renal injury, bilateral renal and ureteral calculi, chronic pyelonephritis, vesical calculus, benign hypertrophy of the prostate, tumour of the bladder and urethral trauma. 5 c.c. of sterile isotonic solution of congo red is given intravenously and this injection may be repeated if necessary. The authors note that the drug is of little or no value in actual blood disease and they have used it with success as a prophylactic agent in prostatectomy and transurethral resection. The drug is reported to be of great success also in severe epistaxis and bleeding after tooth extraction. The authors did not find any ill-effects after its use.

Zinc Ionization in Nasal Allergy

BERNHEIMER (*Journ. Amer. Med. Asson.*, June 6, 1936, page 1980) reports on the treatment of 25 cases of hyperæsthetic rhinitis with zinc ionization and observes that in 20 of these cases the treatment was useless having no impression on the course of the disease and in 5 there was cessation of symptoms for varying periods upto 1 year. In another series of 20 cases of hyperæsthetic rhinitis the author used the application of trichlor acetic acid or phenol to the anterior tips and medial and lateral surfaces of both inferior turbinates and both sides of anterior septal wall and the result was more satisfactory with much longer periods of remission. The author then reports on the failure of both zinc ionization and escharotic applications in his cases of hay fever and cites two complications following ionization treatment, *viz.*, anosmia in one and sphenopalatine neuritis in another though he had seen none in patients treated with escharotic agents.

Control of Bleeding in Nose and Throat Operation

BROWN (*Med. Journ. Australia*, March 14, 1936, page 339) in dealing with the problem of control of bleeding in nose and throat operations discusses the physiology of blood coagulation and advises the following steps to be taken to fill up

deficiency in blood coagulation, *viz.*, (1) introduction of normal prothrombin from some foreign source, (2) introduction of any of the other coagulative elements and (3) introduction of some foreign substance to stimulate more rapid coagulation. The author then deals with the importance of noting the blood clotting time and the bleeding time. The author advises to remove the clot as bleeding not infrequently comes from beneath the clot and to place the patient in an upright position after the operation as the latter diminishes the patient's blood pressure, increases the tone of the cut ends of the vessels and diminishes the pressure of venous blood above the bleeding points.

Problems in the Diagnosis of Cancer of the Stomach

DELARIO (*Amer. Journ. Cancer*, June, 1936, page 334) in dealing with problems in the diagnosis of gastric cancer observes that 50% of them occur in the pyloric end, 30% in the pars media, and 20% in the cardiac end and that it is in the last group that greatest number of mistakes are made so far as the roentgenologists are concerned though practically all examples of gastric cancer which are benefited by roentgen therapy occur here. He further observes that patients with anaemia, loss of weight and gross blood in the stomach even with no gross x-ray findings probably have cancer of the cardiac end and if the rugae technique be of no help these patients should have the benefit of laparotomy. The author observes that in their experience benign gastric ulcers have never undergone malignant change and that in presence of painless jaundice the possibility of hepatic cancer metastatic from the stomach should be borne in mind.

OBSTETRICS AND GYNÆCOLOGY

Haemoglobinuria as a Symptom of Ruptured Ectopic Pregnancy

POMMERENKE (*Amer. Journ. Obst. Gynec.*, April, 1936, page 650) observes that haemoglobin in the urine is a common finding in ruptured ectopic pregnancy with intra-abdominal haemorrhage and in the differential diagnosis its presence (by chemical and spectroscopic tests) should be determined and evaluated in the presenting syndrome.

A Record of 26 Cases of Rupture of the Uterus

SHELDON (*Amer. Journ. Obst. Gynec.*, March, 1936, page 554) reports that of 47,554 deliveries at the Boston Lying-In-Hospital rupture of the uterus occurred is 26. 65% of the ruptures were due to trauma during operative delivery through the pelvis. In 12 of 17 cases of traumatic rupture internal podalic version was the ultimate type of delivery. Of 9 spontaneous ruptures 5 occurred in cases who had previous caesarian section. Multiparity is an etiological factor and only 2 primigravidas had spontaneous ruptures. The maternal mortality was 42·3% and the foetal mortality 82%. Treatment of choice is hysterectomy immediately after rupture.

The Role of Anaesthesia in the Production of Asphyxia Neonatorum

EASTMAN (*Amer. Journ. Obst. Gynec.*, March, 1936, page 563) summarises his observations on anæsthesias producing asphyxia neonatorum:— (1) " Chloroform administered to mother at the time of delivery has no demonstrable effect on the oxygen saturation of foetal blood, but due to its toxic effect on the mother it is not to be recommended as an obstetrical anæsthesia. (2) Ether anaesthesia produces a slight depression of the oxygen saturation of foetal blood, but this is not of sufficient degree to injure the foetus through anoxaemia. (3) Nitrous oxide oxygen mixtures, administered to mother in proportion of 85:15 or weaker and for period of less than 5 minutes, regularly causes moderate degrees of foetal anoxaemias but the normal, full term infant is apparently not harmed. (4) When nitrous oxide oxygen is given in concentration of 9:10 or stronger over periods which exceed 5 minutes marked degree of foetal anoxaemia are produced in about one baby out of 3 and occasionally profound asphyxia neonatorum results."

PATHOLOGY AND BACTERIOLOGY

Sodium Citrate—A Spirochaeticide

LEADINGHAM (*Journ. Lab. and Clin. Med.*, June, 1936, page 922) observes that a 2% solution of sodium citrate (which is used in the laboratory to prevent coagulation of coverslip preparations of

blood and exudates) causes immediate cessation of movement and ultimate dissolution of the spirochaetes taken from a laryngeal lesion of Vincent's angina. The author found this to be true on repeated examinations. He further observes that when the solution was applied topically on the ulcer it destroyed the bacteria so completely that further swab examinations were negative. The author concludes by saying that " should this drug find a place in the local and intravenous treatment of spirochætal infections, it might replace much more expensive and harmful therapy."

Blood in Alkalosis

COPE (*Clinical Science*, Vol. 2: No. 3, page 287) reports on the study of several cases of alkalosis produced as a result of treatment of gastric ulcer with alkalies and in summarising the findings regarding the inorganic basic constituents of the blood of such patients observes that the serum total base, the serum calcium and the serum magnesium are all definitely raised above normal limits.

The Role of Malaria in the Causation of Cirrhosis of the Liver

TIRUMURTI AND RAO (*Ind. Journ. Med. Res.*, July, 1936, page 149) in reporting on the critical investigation of morbid histological changes in chronic malarial livers by using different staining methods observe that there is no loss of lobular pattern and that there is absence of marked fibrosis as shown by silver impregnation of the liver reticulum, and conclude that malaria *per se* is not a direct cause of cirrhosis of the liver.

Bone Formation in Laparotomy Scars

VOECKER (*Zentralbl. f. Chir.*, March 14, 1936, page 626) reviews the literature on the subject and observes that of the 70 cases reported majority have been found in median incisions above the navel in males with peptic ulcer. The bone formed may be in single or several pieces and may be of different shapes and sizes. The author then reports a case which developed an elliptical ring of bone measuring 4 by 8 cm. in 2 year's time from a scar following an operation for perforated duodenal ulcer. Bone formation may follow septic or aseptic healing and trauma, formation of haematoma, necrobiosis and unknown factors of metabolic origin usually helps in its formation.

Bacteriology of Puerperal Sepsis

SCHWARTZ AND BROWN (*Amer. Journ. Obst. and Gynaec.*, March, 1936, page 379) report on the results of their study on puerperal infection for a period of ten years. They observe that 228 cases of puerperal infection occurred in 13,237 deliveries and of these 216 were cases of acute endometritis. Aerobic bacteria were found out on culture in 35 cases and anærobic in 128 cases and mixed in 52, and of the anærobic bacteria streptococci were predominant. These strains were also found in vaginal smears in 40% of the patients at term and the authors observe that infection took place chiefly after prolonged labour causing damage of the parturient canal. There was nearly 50% reduction of sepsis by prophylactic use of antiseptic vaginal instillation of 1% neutral acriflavin in glycerine. They corroborate Schottmuller's observations that the danger of puerperal infection comes more from within than from without.

PEDIATRICS

Recovery from Influenzal Meningitis

SPEKTER (*Amer. Journ. Dis. Child.*, March, 1936, page 614) reports of a case of influenzal meningitis with recovery. The patient was a girl of 6 admitted to the hospital on the 5th day of an attack of meningitis. Lumbar puncture showed turbid fluid containing polymorphonuclear leucocytes and questionable organisms and she was given 15 cc. of antimeningococcus serum after each of the first 2 punctures. The culture of the C. S. fluid showed the growth of B. influenzae and she was given anti-influenzal serum by intrathecal, intracisternal and intramuscular routes. She received in total 215 c.c. of anti-influenzal serum and 74 cc. of human serum and she got round without any complication.

Pneumonia in Children— A Survey of one thousand cases with attempted follow-up

KOHN AND WEINER (*Amer. Journ. Dis. Child.*, May, 1936, page 1095 report on the study of 1,000 cases of primary non-tuberculous pneumonia in children. They observe that the mortality

was 20% and of the children who left the hospital alive 40% had positive physical findings in the lungs and about 57% had roentgen evidence of infiltration. Of the 80 cases who were kept under observation after discharge from the hospital 36 completely got rid of those evidences of infiltration etc., in less than 6 months. The severity of the initial illness was no criterion of the persistence of the physical signs revealed by roentgen examination. Empyema, peritonitis and involvement of the kidneys and brain were the main complications.

Atropine Fever

ALANCON (*Br. Journ. Child. Dis.* April—June, 1936, page 135) reports to the Societe de Pediatrie that 9 out of 125 infants when treated with atropine for dyspepsia developed sudden rise of temperature with generalised cutaneous hyperaemia, dryness of the mucous membrane of the mouth, restlessness and sometimes spasm of the larynx. These phenomena are explained by the stimulating action of atropine on the sympathetic and vagus, the sympathetic action stimulating the thyroid secretion and producing accelerated metabolism and rise of temperature and the vague action producing hyperaemia of the skin and suppression of sweat.

Artificial Pneumothorax in Children

ROLESTON (*Br. Journ. Child. Dis.*, April—June, 1936, page 93) in reporting 5 cases of pulmonary tuberculosis treated with artificial pneumothorax observes that in unilateral involvement A. P. is as applicable in children as in adults and the results thereof are quite encouraging and that the tendency to effiusion seems less in children than in patients over 20 who seldom escape effusion of some degree.

Prognosis of Tetanus

COLE (*Lancet*, II. 1935, page 957) says that in local tetanus where the incubation period is long the prognosis is good. In generalised tetanus where there is the general rigidity, and where reflex spasms do not occur and the incubation period is long, the prognosis is good, although the disease may last for weeks. In cases in which the reflex spasms come on within 24 hours the prognosis is bad. In cephalic tetanus convulsions and cranial nerve palsies may occur and the outlook is serious. The severity of the disease depends upon the amount of toxin. With small amounts only local tetanus occurs; with large amounts reflex spasms supervene. The larger and dirtier the wound the worse the prognosis. If the wound is a penetrating one or covered by a scab B. tetani can flourish anaerobically and more toxin is produced. The general health and physique are of great importance in prognosis. In infants and young children the prognosis is bad. The longer the incubation period the better the prognosis. Cases with incubation periods of less than 5 days hardly ever recover. The longer the period between stiffness of the jaws and the first generalized reflex spasm the better the prognosis. If reflex spasm occur in less than 2 days, there is little hope of recovery. The more thorough the treatment and the more the reflex spasms are controlled the better the outlook. Avertin in doses larger than those used in anaesthesia controls the spasm better than any other drug. The more nourishment the patient takes the sooner is he cured.—(*Br. Journ. Child. Dis.*, April—June, 1936, page 156).

PHYSIOLOGY, BIOCHEMISTRY AND NUTRITION

The Duodenum and the Automatic Control of Gastric Acidity

GRIFFITHS (*Journ. Physiology*, Vol. 87. No. 1, June, 1936) observes that (1) following the experimental introduction of hydrochloric acid directly into human duodenum the secretion of hydrochloric acid by the stomach in response to an alcohol test drink is greatly diminished; (2) that if hydrochloric acid is allowed to flow into the duodenum shortly after gastric secretion has been established by alcohol, gastric secretion of hydrochloric acid is arrested before it has attained its maximum; (3) that in both cases although hydrochloric acid secretion is diminished or inhibited, the neutral chloride and peptic activity of gastric contents show a marked rise. It is suggested that entry into duodenum of acid is an important factor in regulation of gastric acidity and this conception is favoured by clinical experiences. Conditions which obstruct passage of material from stomach are frequently associated with failure of acid regulating mechanism and the post

digestive fall in acidity is delayed or fails (Bolton). It is also confirmed by works of Day and Webster.

The Chemical Transmitter of Motor Impulses

HARRISON AND MCSWINEY (*Journ. Physiology*, Vol. 87, No. 1, June, 1936) write that stimulation of the vagus, the sympathetic trunk and the splanchnic nerves may cause contraction of stomach in spinal cat and the contractions are enhanced after injection of eserine and abolished by atropine. They further note that similar effects were obtained with isolated preparation of fundus of the stomach without mucous membrane and innervated by the vagus and periarterial nerves. It is suggested that both the sympathetic and vagus nerves contain cholinergic fibres (as both also contain adrenergic fibres).

Vitamin C and the Adrenal Control Hormone

SVIRBELY AND KENDAL (*Amer. Journ. Physiology*, Vol. 116, No. 1, June, 1936) observe that adrenalectomised dogs maintained on a diet free from vitamin C did not develop scurvy and that no change in nitrogen metabolism (such as increased urea content of blood and decreased nitrogen output) ordinarily resulted when adrenalectomised dogs, receiving a constant amount of cortin, were maintained on a diet free from vitamin C. The fluctuations in nitrogen metabolism depended only on the amount of cortin injected and not on vitamin C. In contradistinction to previous reports (Lockwood, Hartman) cortin has no effect in delaying the onset of scurvy and ascorbic acid is the main factor in preventing scurvy in guineapigs.

Influence of Liver in Formation and Destruction of Bile Salts

BOLLMAN AND MANN (*Amer. Journ. Physiology*, Vol. 116, No. 1, June, 1936) write that normal dogs excrete traces of bile salts in urine and bile salts are not detectable in blood and that following intravenous injection of bile salts even in large amounts only a small portion appears in the urine and the injected material rapidly disappears from blood showing destruction in the body. They observe that after complete removal of the liver, bile salts are not found in urine and blood; and therefore the liver appears to be the site of formation of bile salts. They further observe that after complete removal of the liver injected bile salts are excreted in the urine and quantitative recovery is possible within 12 hours and the liver thus plays a prominent part also in the destruction of bile salts.

Is a Portion of Pancreatic Secretory Response to a Meal due to Absorption of Digested Food Stuffs?

GRAY, KIM, IVY (*Amer. Journ. Physiology*, Vol. 116, No. 1, June, 1936) tried intravenous injection of various products of digestion, viz., (1) protein-liver extract and Witte's peptone, (2) dextrose, (3) emulsified fat, and conclude that secretin is probably the only humoral agent concerned in secretory response of the pancreas to a meal.

Lesions in Pancreas and Ant. Hypophysis with Fatal Acidosis following Prolonged Intravenous Administration of Glucose in Dogs

JACOBS AND COLWELL (*Amer. Journ. Physiology*, Vol. 116, No. 1, June, 1936) write that prolonged and continuous administration of large amount of glucose by vein to normal dogs results in:— (1) early and sustained improvement in capacity to make use of the administered sugar, failing terminally, (2) remarkable hepatic storage of glycogen; (3) progressive depletion of alkali-reserve resulting ultimately in fatal acidosis; (4) generalised congestive changes in all viscera; (5) haemorrhage into and destruction of the pancreas and anterior pituitary. The authors observe that convincing interpretation of these phenomena is not possible and the formation of excessive amounts of unidentified acidic intermediate products of glucose metabolism is surmised.

MEDICAL NOTES AND NEWS

MEDICAL PRACTITIONERS AND POISONS RULES

A Press communique says:—

It is notified for general information that the rules framed by the local Government in certain notifications issued since 1934 to regulate the dealings of medical practitioners in British Pharmacopoeia poisons have been rescinded to the extent that they apply to qualified medical practitioners. whether registered or not.

A conference which considered these rules at Naini Tal recently advised the Government that the rules, except in their application to practitioners without registrable qualifications, should be cancelled and the Government have decided to accept the recommendation.

The conference recommended further that the question of issuing fresh rules upon the subject, in strict accordance with the rules which apply to medical practitioners in Great Brita'n, should be taken up.

The Government have accepted this recommendation also, and the question will be referred shortly to the medical council for their opinion and advice.

PRIZES FOR THESES

Dr. M.B. Narayan Rao, Managing Editor, Indian Journal of Venereal Diseases, Bombay, announces two more prizes for the best article in each of the subjects mentioned below:

1. "Manwantar Printing Press Prize" of the value of Rs. 100/- for the best thesis on Serology in the Diagnosis and Prognosis of Venereal Diseases.

2. A prize of the value of Rs. 100/- for the best thesis on "Investigations into Lymphogranuloma Inguinale."

The prize articles will be selected from amongst those published in the succeeding six issues of the Indian Journal of Venereal Diseases, September, December '36, and Jan, March, Sept., and December, 1937.

The "Charak Memorial Prize" of the value of Rs. 100/- has been awarded to Dr. D. V. Subba Reddi, M.B.B.S., Medical Registrar, Medical College, Vizagapatam. A consolation prize of the value of Rs. 25/- has been awarded to Dr. H. S. Joshi, L.C.P.S. (Bombay), of Nasik.

Detailed information may be obtained from the Managing Editor, Indian Journal of Venereal Diseases, Bombay.

RECHLORIDATION FOR POSTOPERATIVE TOXEMIA

Recent studies have revealed the existence of a postoperative toxemia which may not only be checked in its incipient stages but the development of which may be prevented by the administration of sodium chloride solution in high concentration. In its mildest from, such a toxemia presents itself in the first few days following operations as a feeling of exhaustion, slight evidences of intestinal paresis, decreased urinary output and lowered blood pressure. If the symptoms increase, the malaise and weakness become more marked, the tongue is dry, the blood pressure drops more, the pulse increases in rapidity, hiccup appears and the oliguria may progress to almost complete anuria.

The evolution of this more severe clinical picture varies greatly. The symptoms may recede spontaneously and slowly or a more serious termination may ensue either with a single symptom or with a combination of symptoms. Under the former heading one may encounter cases of (a) coma with anuria and Cheyne-Stokes respiration, (b) uncontrollable vomiting resembling an actue ileum, (c) marked irregular elevations of temperature or (d) a case in which the signs of cardio-vascular disturbances predominate. A fatal outcome may ensue gradually or occur with lightning-like rapidity. As the result of the work of Chabanier and Lobo-O'Nell, Levy, Legueu and Fey and of Duval, the mechanism of such a postoperative toxemia has been greatly cleared up. The following working hypotheses, the majority of which have been proved by laboratory studies, have been set up:

1. Disturbance of nitrogenous metabolism.
2. Disturbance of chloride metabolism.
3. Lack of acid-base equilibrium.

The hypochloremia does not express itself by any important sign clinically. Following operation, a patient may present a marked hypochloremia and yet seem to be making an uneventful recovery. On the other hand there may be only a slight hypochloremia and grave symptoms of toxemia may be present. The increase in residual nitrogen seems to dominate the time of appearance and degree of severity of the symptoms of toxemia (postoperative).

In a postoperative case in which either the milder or more severe symptoms appear as previously mentioned, a chemical analysis of the blood and urine should be made immediately. The blood examination must include percentage of urea, plasma and globular chloride content and glycemia. The urine examination embraces percentage of urea, chlorides and acetone. All the results can be obtained within two hours. The marked decrease or complete disappearance of chlorides in the urine gives one the best idea of the degree to which chlorides have been withdrawn from the blood and tissues to be lodged in the operative zone. One should begin the treatment by the administration very slowly of 20 cc. of a 4 per cent solution of sodium chloride. The total amount of sodium chloride to be injected during the first and following days depends first on the degree of dechloridation. The more marked the diminution of chlorides in the urine, the more sodium chloride should be given. Secondly, the patient whose symptoms of toxemia are very serious requires a larger amount of sodium chloride, i,e, more rechloridation, than a patient with only mild toxemia. Thirdly, the longer the symptoms have lasted, the more sodium chloride is needed. Twenty c.c. is inadequate if all three of these are very marked. Under these circumstances first dose should be from 30 to 40 c.c. of the 4 per cent. solution followed in from eight to twelve hours by a second one of 20 c.c.

The blood and urine should be examined daily and the treatment continued until the blood chlorides and urea have returned to the normal figure, the quantity of urine is greatly increased, and all clinical symptoms have receded. In general, it suffices to give between 20 and 30 Gm. of sodium chloride the first day, between 10 and 20 the second day and between 5 and 10 the third.

The results of this rechloridation treatment have been most gratifying, especially following abdominal operations in general surgery and after prostatectomy (*Jour. A. M. A.*, June 13, 1936, Volume 106, No. 24: page 2080-2081)

A BUREAU OF HUMAN HEREDITY

Some well known men, including Lord Moynihan, Sir Arthur Keith, Sir Grafton Elliot-Smith, Sir E. Gowland Hopkins and Sir Humphry Rolleston, have formed a committee to obtain support for the Bureau of Human Heredity, which was recently set up in London. In a joint letter to the *Times* they ask for 50,000 to carry on the work for five years. They point out that problems of national health have reached a point at which the hereditary element can no longer be neglected. This applied not only to preventing the transmission of defects. It is recognized that methods of cure must vary with the type of constitution of the patient, and here information as to heredity is important. In education, in training and in choice of a career the ascertainment of innate endowments is useful. The imperial Bureau of Plant Genetics at Cambridge and of Animal Genetics at Edinburgh have achieved much by setting up simple machinery for collecting information based on the result of research and making these available for the practical breeder, The Bureau of Human Heredity will follow these models. The signatories say that in these days of international mistrust and animosity it is refreshing to find a field in which representatives of nearly every civilized country are engaged in cooperative work. A scheme for an international clearing house of facts concerning human heredity has been evolved by a small international committee, which has delegated to its British members the task of setting up a bureau in London for the collection and distribution of all authentic information on human genetics. The signatories point out how strange it is that students of fruit, flies or mice have at their command the latest information, while those similarly concerned with man can look nowhere for a complete survey of the knowledge they require. (*Jour. A. M. A.*, June 27, 1936: Vol. 106, No 26: pp. 2247)

SOME PROBLEMS OF PHYSICIANS

The first reunion of the *Corporazione delle professionale delle arti* was recently held in Rome under chairmanship of the head of the government. The

vicepresident of the corporation observed that the free activities of the medical profession come to a standstill whenever new institutions of the nature of the Casse Mutue Sanitarie (centers for medical care of insured workers) are opened. He stated that in order to protect medical practice it is necessary to review the plans of the several branches of medicine to establish restricted and special functions for each different branch and to make compulsory the registration of practitioners in the book for that purpose. The new national medical fees were discussed. The National Syndicate of Physicians suggested a plan of fees in which there is a tendency to standardize the fees of the several provinces and to fix a minimal fee without indicating the maximal charges, excluding from the fee the medical attendance given by specialists, regular professors and university professors. The following are the charges proposed: visits for consultation and care of the patient at his own home, 20 lire ($ 1) if it is only one visit and 15 lire ($ 0·75) if there are several consecutive visits. The charges for visits for consultation and care of the patient at the physician's office will be 15 lire ($ 0·75) for only one visit and 10 lire ($ 0·50 for several consecutive visits. Consultations in emergencies will be charged with 25 lire ($ 1·25) if during the day and 40 lire ($ 2) if during the night. (*Jour. A. M. A.*, June 27, 1936: Vol. 106, No. 26: pp. 2251).

THE NEW MIDWIFERY SERVICE

In the House of Commons Sir Kenneth Wood, minister of health, moved the second reading of the midwives bill, the purpose of which is to establish a service of salaried trained midwives, so that every expectant mother, whatever her circumstances, can obtain a qualified midwife. The bill is also designed to raise the status of the midwifery profession by providing adequate salaries and facilities for further instruction. In the last ten years nearly 2,000 mothers have died annually in childbirth. The failure of maternal mortality to yield hardly a decimal point to sustained attacks has been a matter of national concern, contrasting with the notable results in the case of infant mortality. Last year the government spent over 15,000,000 on maternity and child welfare, yet the maternal mortality was not substantially reduced

The local authorities will control the new service. They will fix the salaries of the midwives employed by them and the fees to be charged. A midwife now practising can either apply for a salaried post or continue her independent practice. On the other hand, she can within three years of the coming into operation of the new scheme surrender her practising certificate and receive as compensation thrice the average of her emoluments for the preceding three years. The local authority can call on old or infirm midwives, unable to perform their duties satisfactorily, to retire, in which case they will receive five times the net value of their practice in the preceding three years. Arrangements are provided to enable midwives to keep abreast of current practice by attending refresher courses. The Government will assist the local authorities by supplying approximately half the total additional cost incurred for the service. But the government grant will vary with the needs of the area from 25 or 30 per cent in the richest areas to 80 per cent. in the poorest.

The local authorities will also be encouraged to develop further their antepartum clinics, which have increased by 20 per cent since 1931. The case of London has been specially considered, as salaried midwives are already provided by the London voluntary hospitals and about 25 per cent of the labours take place in municipal hospitals, where there are antepartum clinics and specialists in all branches of maternity work. The new salaried midwives will have these services at their back and can enlarge their experience by taking duty in the hospitals when not engaged in domiciliary work. (*Jour. A. M. A.*, June 20, 1936: Volume 106, No. 25: pp. 2169)

RESEARCH IN TROPICAL MEDICINE

Fellowships And Appointment.—The Medical Research Council, advised by the Tropical Medical Research Committee which it has recently appointed after consultation with the Colonial Office, makes the following announcement:

Junior Fellowships.—Three junior fellowships are offered immediately, for award to qualified medical men wishing to receive training with a view to careers in research work in tropical medicine. Preference will be given to candidates who have already had preliminary experience of methods of research in some branch of medical science. Subject to satisfactory reports, the fellowships will be tenable for three years. The first year will be spent at a school of tropical medicine; the second in doing research in

the same or some other institution at home; and the third largely in work under direction at some centre in the Tropics. The stipend will be at the rates of £300, £400, and £500 per annum in the successive years, with an additional allowance during service abroad and necessary expenses.

The Council has every reason to believe that men who undergo this training will be eligible and well qualified for various appointments, apart from the prospects of further employment under the scheme now announced.

Senior Fellowships.—In three years' time at least one senior fellowship will be available for candidates who have held the junior fellowships mentioned above. This will be awarded for a further period of three years, carrying stipend at the rate of £600 to £750 per annum, with an additional allowance during service abroad and necessary expenses. The time will be spent mainly in research work in the Tropics.

The Council is also prepared to consider immediate applications for senior fellowships from candidates who have had adequate experience in research work, whether already specially trained in tropical medicine or not.

Permanent Appointments.—The Council further intends to establish in due course, as suitable investigators become available as the result of the fellowships scheme, permanent and pensionable appointments for research work in tropical medicine, including senior posts. Members of this research staff will work partly in the Tropics and partly in institutions at home, to which they will be severally attached. The exact terms of service are still undecided, but they will not be less favourable than those which apply to other Government appointments at home or over-seas for men of similar professional standing.

Inquiries may be addressed to the Secretary, Tropical Medical Research Committee, 38, Old Queen Street, London, S.W.1, with whom applications should be lodged not later than October 1st, 1936.

MEDICAL TRUANTS

Lord Moynihan in his Linacre Lecture reviews the careers of a number of persons who were medical men by profession and occasionally by practice but who became famous in callings other than medical. Thomas Linacre in whose memory the lecture was founded was better known as a classical scholar than a physician. Of medical men who illuminated the field of science are mentioned amongst others the names of Galileo; Galvanis, the physicist; Robert Boyle, the formulator of Boyle's Law; Thomas Young, the famous worker in physical optics; James Hutton who made the first geological maps and Thomas Huxley, champion of evolution. Of truants from medicine to statecraft and politics are described the careers of Sir Starr Jameson of Transvaal 'raid' fame; Viscount Finlay, the Lord chancellor in Lloyd George's first government; Foster who later on was raised to the House of Lords and became Lord Ilkeston; Joseph Guillotine, the French humanitarian who proposed that "for all classes capital punishment should be by decapitation"; Clemenceau, the 'Tiger' and the Saviour of France during the last European war; Sun-Yat-Sen, the first President of the Chinese Republic; General Leonard Wood, the greatest colonial administrator America has yet produced; Sir Hilarion Fernando of Ceylon who later on distinguished himself in works connected with agriculture and economics.

Amongst truants to literature are mentioned the names of Oliver Goldsmith; Schiller; Keats; George Crabbe; Robert Bridges, the Poet Laureate; Thompson, the surgeon and novelist; Conan-Doyle; Oliver Wendel Holmes; Weir-Mitchell; Edward Jenner, the discoverer of vaccination and Ronald Ross of malaria fame, who wrote poetry and mathematical exercises.

Other medical truants included Sir Francis Seymour Haden, the famous etcher; the explorers Mongo Park and Livingstone and Henry Jones, the supreme authority on whist.

The lives of some of these great men as described by the lecturer are most romantic and highly interesting and illustrate the influence of scientific training upon all mental activity. The lecturer concludes by saying that "a scientific training is useful, not only in the work of an arduous and scientific profession, but in all other callings into which by accident or deliberate intention a man may chance to wander."

ASSOCIATION NOTES

DACCA BRANCH

A meeting of the Indian Medical Association (Dacca Branch) was held on 12-8-36 at 7-30 P.M. in the Jagannath Intermediate College premises.

Rai Dr. G. P. Mitra Bahadur presided.

Members Present:—

Drs. S. K. Mitra, N. Banerjee, H. Ganguly, S. C. Roy, L.K. Sen Gupta, S. C. Banerjee, N. N. Guha, K. P. Chakravorty, C. H. Dey, B. L. Roy, M. L. Boral and J. K. Sen Gupta.

1. This meeting places on record its deep sorrow at the sad death of Dr. A. N. Das, a member of the Indian Medical Association. Moved from the chair all standing in silence for one minute.

2. Resolved that Sir Nilratan Sircar, KT. be nominated for the presidentship of the Bengal Provincial Branch for 1936-37 and that—

(a) Dr. B. C. Roy, M.D., M.R.C.P., F.R.C.S.

(b) Rai Dr. G. P. Mitra Bahadur, M.B.

(c) Major J. H. Thomas, I.M.S.

be nominated to be three Vice-Presidents of the Bengal Provincial Branch for 1936-37.

3. Resolved that the following members do form a Sub-Committee to consider Dr. K. S. Ray's letter, dated 20-7-36 regarding unemployment among medical profession:—

Capt. C. C. Das Gupta, I.M.S. (LATE), Drs. J. K. Sen Gupta, H. Ganguly, L. K. Sen, S. C. Roy and N. C. Banerjee.

4. Read Dr. K. S. Ray, Joint Honorary Secretary's letter inviting attention to item No. 5 of the proceedings of the 37th meeting of the Central Council held on 13-6-36 regarding anti-medical council day. It was resolved that this Branch will co-operate if invited by the All-India Licentiates Association to do so.

5. With a vote of thanks to the chair the meeting terminated.

N. BANERJEE,
Jt. Honorary Secretary.

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION

The Tinnevelly District Medical Association held its monthly meeting in the splendid palace of Ramnad on Saturday, the 25th July, 1936 at Courtallam. Members numbering 52 including two lady doctors, gathered from all parts of the District.

After light refreshments the meeting began under the presidentship of Lieut.-Col. T. S. Shastry, I.M.S., at 11 A.M. The Secretary Dr. K. Rama Ayyar, M.B.B.S., read the minutes of the monthly meeting held at Papanasam on 27th June, 1936 which were passed.

Dr. G. Parathasarathy M.B.B.S., demonstrated a case of Congenital Heart Disease (Pulmonary Stenosis).

Dr. A. L. Ananthanarayana Iyer, M.B.B.S., Tenkasi demonstrated cases of Mitral Stenosis, Aortic regurgitation with aneurysm of the aorta, Chancre lip and Myxoedema.

Dr. K. Gopal Rao, L.M. & S., read interesting clinical notes of the following cases, which were operated on by Lieut.-Col. T. S. Shastry, I.M.S., at the Government Head Quarters Hospital, Palamcottah: (1) Lymphosarcoma of axilla, (2) Intussusception—Tumour of Caecum.

Dr. K. Padmanabha Ayyar, L.M.P., of Tenkasi, read an interesting paper on 'Alkalinity of Blood in relation to Health and Diseases'. After discussion, Lieut.-Col. T. S. Shastry, I.M.S., summed up the salient and important features of all the cases, and thanked the Estate Collector of Ramnad for his readiness and kindness in placing the spacious and splendidly furnished palace of Ramnad at the disposal of the Association. Thanks were also offered

to Dr. S. N. Rangachary, and his helpers for organising the most enjoyable function so well.

In the evening, a Health Exhibition was held in the town of Tenkasi.

K. RAMA AYYAR,
M.B.B.S.,
Secretary and Treasurer.

U. P. PROVINCIAL BRANCH

Proceedings of the Working Committee meeting of the Indian Medical Association, U. P. Provincial Branch held on Tuesday the 4th August, 1936 at the Garvie Medical Library, Town Hall, Meerut.

Members Present:—

Dr. S. Halder of Muzaffarnagar, Vice-President in the Chair.
Dr. R. C. Mitter of Meerut.
Dr. Bhupal Singh, Provincial Secretary.
Dr. R. N. Bose. Provincial Jt. Secretary.
Dr. Kunj Behari Lal Verma of Meerut.

I. The proceedings of the last Working Committee meeting were read and confirmed.

II. Accounts for the months of June and July, 1936 were placed. Resolved that the Accounts be passed.

III. Nominations from the branches for the Presidentship of I.M.A., U. P. Provincial Branch for 1936-37 were considered.

17 nominations were received out of which 14 nominations were received in time ie., by the 21-7-36 and all these branches nominated Capt. S. K. Chowdhry of Benares, the other 3 branches nominated as follows, the dates of receipt of their nominations here are noted against them.

Saharanpur Branch nominated Dr. Abdus Samad of Cawnpore, received on 22-7-36.

Mussorie Branch nominated Dr. P. Caul, received on 2-8-36.

Khurja Branch nominated Capt, S. K. Chowdhry of Benares, received on 22-7-36.

(a) Resolved that only nominations received here upto 21-7-36 be considered, Dr. Kunj Behari Lal Verma dissenting.

(b) As Capt. S. K. Chowdhry's name is the only name nominated by the branches whose nominations have been received within time *i.e.*, 21-7-36, resolved unanimously that Capt. S. K. Chowdhry be declared elected President of I. M. A., U. P. Provincial Branch for 1936-37 by this Working Committee under powers vested in this Committee by the Provincial Council by that Council's resolution No. 7 dated 12-10-36.

IV. Resolved that the formation of Mussoorie Branch be noted.

V. Letter from the Secretary, Mussoorie Branch, enclosing a copy of the letter dated nil from the Secretary, All-India Medical Licentiates Association was read.

Resolved that a Sub-committee consisting of the following gentlemen be formed to consider and draft a reply to the copy of letter forwarded by Mussoorie Branch and place the same before the next meeting of this Working Committee:—

1. Dr. Bhupal Singh, Convener.
2. Dr. Kunj Behari Lal Verma.
3. Dr. R. C. Mitter.

VI (a) Letter from the Hony. Jt. Secretary, I.M.A., Calcutta dated 20th July, 1936 concerning unemployment question was read.

Resolved that the Sub-committee appointed in item No. 5. should go through this question too.

(b) The item No. 5 of the X Central Council meeting regarding participating by calling meetings to observe the All-India Anti-medical Council Day on 20th September, 1936 was read.

Resolved that the contents be noted .

With a vote of thanks to the Chair the meeting dispersed.

R. N. BOSE,
CAPT.
Hony. Provincial Jt. Secy.

S. HALDER,
Vice-President,
Chairman.

MEDICAL ASSOCIATIONS, SOCIETIES, ETC.

Medical Associations, Societies, Unions, etc., are invited to make use of this section of the Journal.—EDITOR.

BENGAL OBSTETRIC & GYNÆCOLOGICAL SOCIETY

A general meeting of the newly started "Bengal Obstetric and Gynæcological Society" was held on the 25th July, 1936, at the Indian Medical Association Hall. A large number of medical men interested in Obstetrics, Gynecology and allied subjects were present.

Dr. S. K. Lahiri was voted to the chair.

At this meeting the constitution of the Association drawn up by the sub-committee formed at the inaugural meeting was adopted, with slight modification.

Members were enrolled at this meeting and the following office-bearers and members of the Executive Committee were elected:—

President—Dr. S. K. Lahiri.
Vice-Presidents—1. Dr. S. N. Bagchi.
2. ,, J. C. Chatterjee.
Honorary Treasurer—Dr. Subodh Mitra.
Jt. Hony. Secretaries—1. Dr. M. Sarkar.
2. Dr. A. K. Acharya.

Committee Members:

1. Dr. Alice Headwards,
2. ,, P. Das,
3. ,, J. Chakraverti,
4. ,, P. Sur,
5. ,, B. Sen Gupta.

All medical men interested in Obstetrics, Gynæcology and allied subjects are cordially invited to become members of the Society. Further information will be gladly supplied by the Hony. Secretaries, Bengal Obstetric and Gynæcological Society. 67, Dharmatala Street, Calcutta.

A. K. ACHARYA,
Jt. Honorary Secretary.

ROYAL SOCIETY OF TROPICAL MEDICINE & HYGIENE

A meeting of the Royal Society of Tropical Medicine and Hygiene was held on 21st May with the president Sir Arthur Bagshawe in the chair. The subject for discussion was "Intestinal Absorption in the Steatorrhœas".

TROPICAL SPRUE

Dr. Hamilton Fairley spoke on "Tropical Sprue, with Special Reference to Intestinal Absorption". The distinctive features of tropical sprue as mentioned by him are (1) steatorrhœa 80% of the cases showing a total fat value exceeding 25% of the dried fæces on a single examination, (2) hypocalcæmia with blood phosphorus remaining 1·6 to 4 mg. per 100 c.cm., precluding parathyroid deficiency as basis of the hypocalcæmia; (3) flat oval glucose tolerance curves associated with high intravenous curves showing defective glucose absorption; (4) anæmia—occurring late, mostly of macrocytic type but may be of microcytic type due to some intercurrent disease. The oral administration of liver extract (½lb. whole liver daily) gave excellent results when it was combined with a diet of high protein, low carbohydrate and low fat values and this suggested that "defective production of hæmopoietic principle rather than defective absorption underlay the macrocytic anaemia". Dr. Fairley contends that all these clinical signs and symptoms of tropical sprue can be explained if disease is taken as a metabolic breakdown of the gastrointestinal tract characterised by defective absorption of fat or carbohydrate or both with or without defective secretion of Castle's intrinsic factor and that avitaminosis is only secondary and produced either by malabsorption or by sticking to a too restricted diet to effect cure. The speaker concluded by observing that "the real enigma was geographical distribution and whoever solved that prob-

lem would have solved the etiology of tropical sprue".

Coeliac Disease

Dr. C. Wallace spoke on "Intestinal Absorption in Cœliac Disease with Some Remarks on the Effect of Liver Extract upon Carbohydrate Metabolism." He first dealt with the deficiencies in Cœliac disease, *viz.*, of those related to fat, protein, vitamines A. B. C. D., calcium and phosporus, iron and Castle's anti-anæmic factor. He then discussed on carbohydrate absorption and gave the three explanations put forward for the flat oval glucose tolerance curves found in cœliac disease, *viz.*, (1) endocrine disturbance, (2) hypertolerance for glucose based on high carbohydrate intake, and (3) defective absorption and supported evidences in favour of the last theory. He then showed some intravenous glucose tolerance curves taken on patients suffering from cœliac diseases, chronic intestinal indigestion with steatorrhœa, abdominal tuberculosis, cirrhosis liver, before and after treatment with Campolon or Anahemin by injection, or liquid extract of liver by mouth. These showed that total liver extracts contained a factor possibly identical with Himsworth's "insulin-kinase, which was capable of improving the glucose tolerance or the sensitivity to insulin where this had been impaired by carbohydrate deprivation or by liver disease, sometimes to the great clinical benefit of the patient." The speaker observed that this factor was not identical with Castle's anti-anæmic factor.

THE BOMBAY OBSTETRIC AND GYNAECOLOGICAL SOCIETY

Variation of Uterine Action in Labour is the subject selected for the competitive thesis for the Dr. Herculano DeSa's Silver Jubilee prize to be awarded by the Society for the year 1936-37:

Terms Of The Prize

1. The Prize is open to all medical persons registered by the Bombay Medical Council, who shall not be of more than five years standing.

2. The thesis must be sent in five typed copies so as to reach the Hon. Secretaries, Bombay Obstetric and Gynaecological Society, Raj Bhuvan, Sandhurst Road, Bombay on or before the 15th March 1937.

3. The thesis should be designated by a motto instead of the writer's name and should be accompanied by a sealed cover containing the name of the competitor and his post office address. The name of the Prize, the year of competition, the subject of the thesis and the writer's motto should be superscribed on the Cover.

4. No study, essay or thesis that has been published in the medical press or elsewhere shall be considered eligible for the Prize.

5. The accepted thesis shall be the property of the Bombay Obstetric & Gynaecological Society.

6. All the theses shall be returned if not accepted provided the return postage expenses are paid in advance by the writer.

7. In the award of the Prize to the successful candidate, the decision of the committee shall be final.

8. The Prize shall be in the form of cash money and a certificate. The amount shall be approximately Rs. 125/-

9. The Society may request the successful competitor to read his thesis at any of their meetings

10. The Prize shall be awarded at the Society's annual meeting.

THE MADURA MEDICAL ASSOCIATION

The monthly meeting of the Association was held at the Government Hospital, Madura, on the 27th June, 1936, at 5-30 p.m. Dr. A. C. Pariera, L.M.S., presided. Only 13 members attended the meeting.

After reading the Minutes of the previous meeting, the General Body passed the following Resolution and authorised the Secretary to send a copy of the Resolution to the Local President, The King George Memorial Fund, Madura.

"The Madura Medical Association strongly recommends that a major portion of the collections from this District for the King George Memorial Fund be retained in the District for starting a *Cancer Clinic* in the Head Quarters Hospital, Madura."

Dr. A. Narayana Menon, M.B.B.S.., Honorary Venereologist, Government Hospital, Madura, read

a paper on "A few salient points in the Treatment of Syphilis."

The necessity for early and thorough treatment of all cases of syphilis; the use of Tryparsamide and Pyrotherapy in neurosyphilis, the need for periodical serological examinations, the necessity for educating the patients to realise the advantages of early treatment, were the salient points mentioned by the lecturer.

After discussion, the meeting terminated at 7 p.m.

XIII ALL-INDIA MEDICAL CONFERENCE

The following communique has been issued by the Jt. Honorary Secretary, Indian Medical Association:—

The XIII All-India Medical Conference will be held at Karachi in December next during the Christmas holidays under the auspices of the Indian Medical Association, the exact date of the Conference will be announced later on. In order to make the Conference thoroughly representative of the medical profession of India it is requested that all members of the profession join the Conference and take part in the deliberations.

As usual a Medical Exhibition will be held in connection with the Conference. As the Conference will be attended by a large number of medical men from all parts of India it will afford an excellent opportunity to dealers and manufacturers of medical requisites to exhibit their products before the delegates. Those who are willing to take stalls at the Exhibition will please write for particulars to the Organising Secretary, XIII All-India Medical Conference, Said Manzil, Bunder Road, Karachi.

There will be a Scientific Section at which original papers on research work, new methods of diagnosis and treatment of diseases etc. will be read and discussed. Those who intend to contribute to the Scientific Section are requested to send copies of summaries of their papers to the Joint Honorary Secretary, Indian Medical Association, 67, Dharmatala Street, Calcutta, before the 15th November, 1936.

Subjects concerning the vital interest of the medical profession in India e.g., rural medical relief, Health Insurance Scheme, Indian Medical Council Bill etc., will be discussed at the Conference. Branches desiring to move resolutions at the Conference are requested to send advance copies of their resolutions to the Secretary at the Headquarters of the Association, 67, Dharmatala Street, Calcutta, sending at the same time a copy of the resolution to the Secretary, Reception Committee, XIII All-India Medical Conference, Karachi.

As usual the next Annual General Meeting of the Association will be held at Karachi during the session of the Conference. Attention of the branches is drawn to the new rules regarding submission of resolutions by branches at the Annual Meeting, as adopted by the Central Council at its last meeting, reproduced in the August issue of the Journal. Branches desiring to move any resolutions at the Annual General Meeting are requested to send copies of their resolutions in advance to the Joint Honorary Secretary, Indian Medical Association, Calcutta, in proper time.

BOOK REVIEWS

The Practitioners Library of Medicine and Surgery, Vol. VIII (Therapeutics), Edited by George Blumer and Albert J. Sullivan, Published by D. Appleton-Century Co., New York and London. Royal Octavo, 1031 pages with illustrations. Price (for complete set of 12 volumes and General Index) Rs. 485/-. Available from Butterworth & Co. (India), Ltd., Calcutta.

This volume of the Practitioners Library of Medicine and Surgery deals with therapeutics. The contents are first divided into two main parts: (i) General therapeutics and (ii) Special therapeutics, each of them is further sub-divided into fifteen and sixteen chapters respectively. The subject of general therapeutics opens with a chapter on the general management of bed-ridden, convalescent and ambulant patients. The succeeding chapters deal with dietetic management, hydrotherapy, heliotherapy, physiotherapy and electrotherapy. A chapter has also been included in psychotherapy which is so essential for the proper management of not only the neurotic type but also of other types of patients. All these chapters are full of the most up-to-date information, useful both to the general medical practitioners as well as to the specialists.

The therapeutic management, according to the authors, should include (1) remedial treatment, (2) physical measures, (3) dietetic measures, (4) supportive treatment, (5) palliative treatment, and last but not the least (6) general hygienic measures. We would draw the attention of the readers of the book to the importance of such a conception of therapeutics on the management of their cases. In the chapter on diet, it is interesting to note that cooked eggs are said to be more digestible than raw ones. In the chapter on climatology, it has been said that the climatic treatment is only a part and often quite a small part of the patients' treatment. The reviewer only wished that this view was widely well-known. In that case it would have saved many patients from unnecessary and heavy expenses.

Under the section of special therapeutics, the treatment has been described in detail of various diseases which have been grouped according to the systems affected. During a close study of this portion, many old therapeutic beliefs will be shattered. Not infrequently one comes across statements such as: Poultices and cupping are of doubtful value in the relief of pain of pleurisy in pneumonia; haemorrhage in typhoid fever is not due to altered blood coagulation and hence use of thromboplastic preparations, serum and calcium may be dispensed with. For the cardiac conditions in typhoid fever digitalis should be avoided except in aged patients with pre-existing heart disease and congestive failure. In the treatment of cerebrospinal meningitis the injection of a chemical disinfectant into the subarachnoid space is not justified under any condition. In the treatment of haemoptysis the use of cracked ice and ice bag over the chest should be discarded. The internal administration of calcium in pulmonary tuberculosis is probably as useless as it is harmless. In the therapy of gastric disease the oral administration of digestive ferments such as pepsin and pancreatin are not of proven value.

In the coli infections of the urinary tract vaccines are of doubtful value. Thus the methods of treatment advocated in this book have a rational basis and not merely the sanction of use. The authors deserve our heartiest congratulations for the critical and lucid manner in which they have dealt with the subject of therapeutics.

The reviewer has no hesitation in commending this volume to the attention of the general medical practitioners and of the specialists.

J. C. B.

Allergy of the Nose and Paranasal Sinuses, By French K. Hansel, Published by C. V. Mosby Company, St. Louis, 1936. Super Royal Octavo, 820 pages with 58 text illustrations and 3 colour plates.

In recent years, Allergy and its various manifestations have formed the subject-matter for a good deal of clinical and laboratory research. Although the nasal factor in asthma has attracted the attention of medical men all over the world a collective coherent idea of its corelation to allergy in general and asthma in particular have not yet been embodied in the usual text-books. The book under

review serves to eliminate this difficulty. It deals exhaustively first with the physiology of the nose and here many new points have been brought to light and prominence which formerly escaped our attention. It next deals with allergy in general, where its biology, pathology and biochemistry and histopathology have been incorporated in great detail, and these chapters will, no doubt, be a very useful study to the general practitioners who are not so much interested in the nasal and paranasal manifestation of allergy. As a matter of fact, as one goes through the pages of this book one feels that it is more a general discourse on allergy although the chapters dealing with the speciality embodies all the up-to-date knowledge on the subject. The chapters have been carefully arranged to keep a sustained and coherent idea and the bibliography at the end of each chapter is one of the most important feature of the book which will be of great help to those carrying on research in allergy and asthma. The illustrations are carefully selected. There is no doubt that the book will be heartily welcomed by the members of the medical profession.

K. K. G.

Thirteenth Annual Report of the Nowrosjee Wadia Maternity Hospital, Bombay, Demy Octavo, 65 pages.

The report gives a record of the excellent work done by the hospital during 1935. The number of confinements in the hospital during the year were no less than 4,654 which shows in no uncertain way, the degree of public confidence deservedly enjoyed by the hospital. Of this total almost 70% are mill hands who badly want hospital care. No maternity hospital to-day is complete without an efficient ante-natal clinic. One of he most reassuring features in the report is the history of the steady growth of the department of the hospital. 1,490 cases received ante-natal care during the year, a figure that should speak for itself to anyone who has any experience of maternity work in this country. It is to be hoped that this steady growth will be maintained till the day when all hospital confinement cases will come through the ante-natal clinic.

Naturally there would be one or two points which falls short of the ideal. The puerperal morbidity, 605 out of 4959 confinements (=30%) would seem to be high even allowing for the unhygienic surroundings from which a larger proportion of the patients come. It is only fair however to point out that only little more than half of these are from genital and operative causes.

There were 52 cases of placenta previa during the year of which 6 were complete; of these 6 cases in 3 the mother and baby were both lost. Caesarian section was not done in any case of placenta previa. One wonders if this is not too conservative a view.

The general impression however one is left with after a perusal of the report is that the hospital is doing very sound work in giving help where it is most needed. The anthorities are to be congratulated for the efficient administration of the hospital for yet another year.

A. K. A.

A Text-Book of Midwifery in the Tropics, by V. B. Green-Armitage and P. C. Dutta, Published by Butterworth & Co. (India), Ltd., 2nd Edition, 1936. Demy Octavo, 447 pages with illustrations. Price Rs. 8/-.

The fact that it has got into its 2nd edition in a short time is proof positive of the deserved popularity of this excellent book. Our students so far have depended almost entirely on Western text-books. One does not suggest that the fundamental principles of medicine vary in different countries and as such text books by acknowledged masters of the subject should prove to be the most useful to the students. But at the same time it is equally true that for successful practical application of universally accepted general principles the student has to learn to corelate these principles with the conditions under which he has to work—the peculiar difficulties and problems that he is to meet daily in his practice of the healing art. Herein lies the value of the book. The subject is dealt with from an Indian standpoint based mainly on the personal experiences of the authors which in itself is a desirable feature in any medical text-book.

The chapters that should prove most useful are the ones on "Diseases associated with pregnancy." Many a young practitioner will fall back on this mass of information collected from personal experience, when he comes across a similar case. For obvious reasons these conditions are not as fully dealt with in the usual text-books.

Another commendable point in the book is the fact that detailed dietaries are drawn from usual Indian foodstuffs.

The opinions expressed in a book of this nature must of necessity be dogmatic in certain respects, but if that stimulates thought and supplies incentive for further work, it will have served an useful purpose.

Perhaps a little more space might have been given to the toxaemias of pregnancy which unfortunately is so common in India. For instance only the bare principles of the Dublin School treatment of eclampsia are given, but where, according to Solomons himself success depends on strict adherence to detail.

The authors themselves offer an explanation for paucity of diagrams in the book but one feels that the point has been slightly overstretched and that a few more figures would be a welcome addition in a future edition.

One unhesitatingly recommends the book to anybody who has anything to do with obstetrics in India.

A. K. A.

OBITUARY

Lt.-Col. R. KNOWLES, I.M.S.

Lt.-Col. R. Knowles, I.M.S., C.I.E., Acting Director of the Calcutta School of Tropical Medicine, died at the age of 53 of heart failure following gastric haemorrhage on the 3rd of August, 1936 at the Carmichæl Hospital for Tropical Diseases, Calcutta.

Lt.-Col. Knowles joined the I.M.S. in 1908. He became the Assistant Director of the Pasteur Institute, Kasauli but later was appointed to the Bacteriological Department. He joined the Bombay Bacteriological Laboratory in 1916 and subsequently became the Acting Director of the Pasteur Institute at Shillong. He was appointed Professor of Pathology at the Calcutta Medical College and was in 1920 on special duty as Secretary, School of Tropical Medicine. After this he devoted his energies entirely to the cause of the School of Tropical Medicine, and worked ceaselessly to raise it to its present standard.

He is the author of a book entitled *Medical Protozoology* and other publications.

ACKNOWLEDGMENT

Added to the List of Exchange (Foreign)

1. Edinburgh Medical Journal.
2. New Orleans Medical and Surgical Journal.
3. Annales De Dermatologie Et De Syphiligraphie.

Book received for Review

1. An Epitome of Obstetrical Diagnosis and Treatment in General Practice, Vols. 1 & 2, 2nd Edn. by Bethel Solomons, M.D., M.B., B.Ch., published by John Bale, Sons and Danielsson, Ltd., Oxford House, 83, 85, 87, 89, 91, Gt. Titchfield Street, London, W.1.